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MENTAL DISORDERS ASSOCIATED WITH PREGNANCY AND THE PUERPERIUM*

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IN an attempt to determine the existence of a psychosis due to etiologic factors found only during pregnancy, labor and the post-partum period, many valuable and interesting reports of cases have been published. In 1847, James Macdonald[1] made a comprehensive study of puerperal insanity at Bloomingdale Hospital (now New York Hospital, Westchester Division). He included the mental disorders occurring during pregnancy, parturition and lactation, and classified his 66 cases diagnostically as mania, monomania and dementia.

C. F. MacDonald,[2] in 1899, recognized three distinct types of insanity occurring during the puerperal period, namely, puerperal mania, puerperal melancholia and puerperal dementia — the last, however, being rare as a primary affection.

Jones,[3] in an article on puerperal insanity published in 1903, used a more modern classification of these mental disorders:

> (1) that which occurs during pregnancy; (2) that from the date of parturition to six weeks after confinement, which . . . we shall refer to technically as puerperal insanity; (3) insanity occurring during lactation and dating from six weeks after confinement. . . . This classification is more convenient than accurate. . . . Insanity is and ever will be, the product of two factors, stress and heredity.

Kilpatrick and Tiebout,[4] in a study of 72 cases admitted to Bloomingdale Hospital in the years 1911–1923, found that diagnostically their cases fitted under four major headings: delirium, manic-depressive psychosis, schizophrenia and psychoneurosis. As a result of this study, they concluded that childbearing apparently presents many problems of a physical and psychical nature that play a definite role in the production of psychoses. These problems of themselves, however, do not determine the type of reaction but merely serve as exciting or precipitating agents in the onset of the illness.

In 1926, Strecker and Ebaugh[5] presented an article on psychoses occurring during the puerperium. In their study of 50 patients, the diagnoses reported were manic-depressive psychosis (18 cases), toxic-exhaustive psychosis (17 cases), dementia praecox (13 cases), paresis (1 case) and psychoneurosis with psychotic symptoms (1 case).

Saunders,[6] in 1929, reported observation on 75 women admitted to the Sheppard and Enoch Pratt Hospital who developed mental illness coincident to childbirth. This study included only of psychosis before the end of the first month after those patients who gave a history of symptoms delivery. The belief was expressed that a psychosis in the puerperal period may be an isolated experience in the life of a woman; on the other hand, there may be repeated attacks of mental illness with other children, or there may be psychoses at other times.

In 1937, Karnosh and Hope,[7] in their study of 231 cases selected from the records of several hospitals, concluded that the stormiest psychoses associated with childbearing develop in the period immediately after delivery, and that the only consistent or characteristic element in true post-partum psychosis is delirium, which appears in varying intensity.

Smalldon,[8] in 1940, in a careful study of 220 cases admitted to the New York Hospital, Westchester Division, in the years 1922–1938, gives a brief review of the changing opinion of the medical profession regarding the psychotic reaction associated with pregnancy and childbirth. He found that only about 3.6 per cent in the series were classified as deliriums directly attributed to the toxic-exhaustive factors of reproduction. The majority of the patients were found to have manic-depressive psychoses, the depressive type predominating. A lesser number were cases of dementia

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praecox, chiefly of the catatonic and paranoid types. Twenty-eight patients had psychoneuroses, and only 8 of the 220 cases were diagnosed as toxic-exhaustive psychoses. Smalldon emphasizes the difficulty of generalizing regarding the mechanisms of the reactions found in post-partum psychoses. He calls attention to a universally recognized dictum among psychiatrists that "each case of a series of post-partum illnesses must be considered as an individual, non-generalizable problem."

Linn,[9] in a recent study, endeavors to justify the use of the term "postpartum psychosis" as a clinical entity. He obtained his figures from several large centers as shown in his valuable contribution to the literature on this subject.

The mental disorders associated with pregnancy and childbirth appear to have little influence on maternal mortality, as shown by a study conducted by the Obstetrical Society of Boston and the Boston Department of Health.[10] No reference to mental disorders is found in this report.

DeLee,[11] who states that psychoses may begin during pregnancy or may be aggravated by it, observes that a bad heredity is found in half the cases of psychoses occurring during pregnancy and the puerperium. He also states that disturbed metabolism is commoner in women whose nervous systems are not equal to the strain of pregnancy and that, without doubt, the strain of labor is too much for many nervous systems. He believes that prolonged painful labor, without infection, explains many cases of puerperal insanity.

Some observers consider the beginning of involution of the reproductive organs and the early part of the process of lactation to have a distinct bearing on the development of puerperal psychoses.

These studies show that until about 1900 most of the textbooks on the subject made a distinction between mental disorders associated with the puerperium and those occurring apart from childbirth. For the last thirty years or more, the trend has been away from the belief that a specific psychosis occurs in the pregnant and post-partum states. The consensus among most pychiatrists is that etiologically the problems of pregnancy and childbirth do not determine any given type of psychosis.

If studies of these puerperal psychoses have little true scientific value, they do make a distinct contribution to the individual psychiatrist, the general practitioner and the specialist in obstetrics. It is to these members of the medical profession that the laity turn for advice concerning childbearing.

With increasing frequency, local physicians ask whether a woman who has had a mental breakdown following the birth of a child should be advised against further childbearing. These, as well as other pertinent questions relating to psychoses occurring during the childbearing period, stimulated a review, and a further analysis, of the cases admitted to Glenside Hospital. In addition to the results of our own observations, we have, as noted above, turned to the published reports of other observers for additional data.

The individual patient who has recovered from a psychosis occurring post partum is deeply concerned about possible future pregnancies. Her husband is also anxious to know whether further childbearing should be avoided. They sincerely wish to avoid future mental attacks for the patient, and they wish to know the hereditary dangers for their children. They want the truth. Unfortunately, it is not possible to give a positive assurance of freedom from future mental breakdown to any inquirer.

The literature on this subject indicates that a woman who has recovered from a post-partum psychosis or a severe psychoneurosis occurring for the first time during her first pregnancy may perhaps safely become pregnant again. As in other mental disorders not associated with childbearing, the constitutional predisposition of the patient, the personality make-up and the precipitating factors are of great consequence to the physician who attempts to give even a tentative prognosis. Physicians generally regard pregnancy and childbirth as rather simple primitive biologic phenomena. According to statistics, the majority of women who bear children suffer very few complications incident to childbirth. It is rather loosely stated that only 1 in 700 to 1200 women reacts by a psychosis during pregnancy or following childbirth.

The present report is based on a study of a group of 45 patients admitted to Glenside Hospital; 43 entered during a twelve-year period, 1930–1941, inclusive; 23 patients were twenty to thirty years old, and 20 were from thirty to forty years of age when the psychosis occurred. One patient was a forty-one-year-old primipara, and another, a multipara, was forty-three years old at the time of her psychosis. The case records show that there were 27 primiparas, including a patient who was six or seven months pregnant when she was admitted to the hospital.

In the group of primiparas, 17 patients were under thirty years of age, 7 were between thirty and thirty-five, 1 was thirty-six, 1 was thirty-seven, and one was forty-one years of age. The family histories in 16 cases were said to have been negative for nervous and mental diseases; the other 3 patients had histories of manic-depressive psychosis before marriage. Of these, 1 patient had a psychotic heredity, 1 had an unstable family environ-

ment, with a definitely alcoholic sibling, and 1 had an irrelevant family history.

In the multiparous group of 18 patients, the youngest was twenty-three years of age, and the oldest was forty-three. Only 1 patient in this group gave a history of a previous psychosis. The family history in 12 cases was negative for nervous and mental diseases.

Religion did not seem to have any particular bearing in the development of mental disorders in this group: there were 17 Protestants, 15 Roman Catholics, 10 Jewesses and 1 Greek Orthodox. In 2 cases, the religion was not stated.

Twenty-seven of all the patients were reported as being mentally and physically well during pregnancy. Eighteen were emotionally upset for various reasons, such as fear and apprehension, irritability and nervousness, mental conflict about premarital pregnancy, hypochondria and poor adjustment to environmental conditions; 2 patients were mildly depressed, 1 was mentally overactive, 1 had had two babies in eighteen months, and 2 definitely did not want a baby and attempted abortion. In 1 case, the patient was worried because her husband did not want a baby. Two multiparous patients became psychotic following induced abortions (in both cases, infection followed the abortion). One of these women had experienced three normal pregnancies and believed that she and her husband could not afford a fourth child. The other had one child and could not afford to have another because her husband's earnings were very small. The former became mildly upset emotionally only in the last month of pregnancy, and the latter became depressed early.

No history or other evidence of alcoholism was found in this group. Narcotics and analgesics used preceding and during labor seemed to have no significant bearing on the development of the psychosis.

The stress of present-day life did not seem to be greater in this group than in the average American family. In several of our cases, "worry over finances" was given as a contributing factor to the mental breakdown. Since the majority of these patients were admitted to a hospital for nervous and mental diseases for the first time during the twelve years subsequent to the great financial crash of 1929, comparisons between this period and the preceding twelve years cannot be drawn. Worry over financial circumstances and excess of physical labor during pregnancy have doubtless always had a deleterious effect on the health of many women. Eighteen patients were considered emotionally unstable before marriage. There were no unmarried mothers, although in 2 cases pregnancy occurred before marriage. In 7 patients, there was a definite psychopathic heredity.

Intelligence was found to be good in the majority of this group, and several patients were of superior intelligence. Thirty-nine patients were born in the United States, 1 in India, 1 in Asia Minor, 2 in Russia, 1 in Canada and 1 in Scotland. In 22 cases, one or both of the patient's parents were born in the United States.

TABLE 1. *Time of Onset of Mental Symptoms.*

ONSET	NO. OF PRIMIPAROUS PATIENTS	NO. OF MULTIPAROUS PATIENTS	TOTAL NO. OF PATIENTS
During pregnancy	7	1	8
1 to 12 days post partum	19	8	27
13 to 21 days post partum	1	5	6
26 days post partum	0	1	1
3 months post partum	0	1	1
4 months post partum	1	1	2
Totals	28	17	45

Table 1 indicates the time of onset of the psychoses, and Table 2 gives the diagnostic classifications. The following case reports are brief abstracts from the records of patients who developed more or less serious mental symptoms.

TABLE 2. *Types of Mental Disorder.*

TYPE OF PATIENT	MANIC-DEPRESSIVE PSYCHOSIS MANIC	MANIC-DEPRESSIVE PSYCHOSIS DEPRESSIVE	TOXIC-EXHAUSTIVE PSYCHOSIS	SCHIZOPHRENIA	PSYCHONEUROSIS	PSYCHOSIS WITH OTHER SOMATIC DISEASE
Multiparas	3	6	5	2	3	0
Primiparas	7	5	9	2	1	0
Not specified	0	0	0	0	1	1
Totals	10	11	14	4	5	1

CASE REPORTS

CASE 3. *Toxic-exhaustive psychosis.* A 25-year-old multipara had an irrelevant family history. Her previous physical health had been good. She was described as "a pleasant child; normal emotional life; not easily led; a calm, reserved young woman who enjoyed fine things." She graduated from high school at the age of 17, worked as a saleswoman for a few years and then married. Her first pregnancy resulted in premature delivery at 6½ months. Her second pregnancy resulted in the birth of a premature infant weighing 1½ pounds. When told of its death a few days later, she became emotionally upset and claimed that she could see her baby. Removed from the hospital to her home on the 10th day, she rapidly developed somatic complaints, suffered from insomnia and was somewhat violent. There were visual hallucinations, feelings of unreality, many somatic complaints and periods of confusion. At the time of admission to Glenside Hospital, there was a pronounced secondary anemia. Although there was some improvement in her condition, the patient was taken home against medical advice after about 5 weeks of hospital care. There was no evidence of syphilitic infection to account for her inability to carry her babies to full term. The patient was readmitted to this hospital

a few years later suffering from insomnia, and it was believed that she was hallucinated. According to her husband, she had made a good recovery from her previous psychosis within a few weeks and then seemed quite normal. But she constantly looked forward to having children and brooded a good deal over her deceased infant. She was again taken to her home against medical advice at the end of 5 weeks. The second psychosis in this case was diagnosed as a catatonic type of schizophrenia.

CASE 4. *Manic-depressive psychosis: manic type.* A 27-year-old primipara with a definitely psychotic heredity and an intelligence above average, at the age of 17 had a manic-depressive psychosis, manic type. On recovery from this illness, she entered a normal school, graduated, and taught school. She was married at 22. Except for the poor heredity and the fact that a psychosis developed about 10 days post partum, her "nervousness and irritability" shown at times throughout her pregnancy would not have been notable. She went through a fairly typical manic episode, which lasted for about 3 months.

CASE 5. *Manic-depressive psychosis: depressive type.* A 39-year-old, well-educated multipara had had no previous mental attacks. The family history was irrelevant. Her last child was born 3½ months before she was admitted to Glenside Hospital. Physical examination revealed a lacerated cervix; otherwise, the general physical condition was good. This patient was "much depressed before, during and subsequent to confinement." Catamenia had returned before we saw her, and "were too frequent, too profuse, and of too long duration during her hospitalization of 4 months and 10 days." There were some delusions of a mild paranoid type. Mental improvement is recorded as "gradual and uninterrupted."

CASE 10. *Psychoneurosis.* This patient was 35 years old when she married the man by whom she had become pregnant. Her anxiety state dated from the time she was certain of her pregnancy. She had always been reluctant to marry because she had an excellent position and enjoyed working. She was described as gay, sociable, high strung and self willed. She enjoyed dancing, and had many friends. There was a strong mother attachment. She got along very well during the first 3 months of pregnancy, and then became depressed and agitated, brooded over having a child, and was fearful. No psychotic symptoms were present during the 30 days the patient was at Glenside Hospital. Improvement was gradual. She gained some insight and became interested, alert and cheerful.

CASE 17. *Toxic-exhaustive psychosis.* A 25-year-old primipara had been married 18 months when her baby was born. Her marital life was "congenial and happy." Her previous health had been good. She was temperamentally cheerful and lively. The family history was irrelevant. During her pregnancy, the patient was "wonderfully well." She was delivered at a hospital. There were no complications. She remained in the hospital for 10 days, did not sleep well, became apprehensive, and heard comments that disturbed her. The patient was transferred to a hospital for mental diseases for a period of observation. On the 20th day post partum, she was admitted to Glenside Hospital. She was confused, disoriented, overtalkative and destructive but not assaultive; there developed a feeling of unreality about her husband and her mother. Later, the patient admitted vague auditory hallucinations. The blood pressure, temperature and laboratory findings remained normal. She was hospitalized for 4½ months and made a good recovery.

CASE 19. *Psychoneurosis.* A 35-year-old multipara, predisposed to emotional upsets, had had hysterical episodes brought on by frustration 7 years previously. She had always had difficulty in adjusting herself to environmental changes. There was a history of asthmatic attacks for many years. Her mother was psychotic. The patient was said to be very industrious, with an intelligence above the average. She was married when 25 years old. Her first child was 10 and the second (an unwanted baby) was 1 year old. The patient "had not been well since the beginning of her last pregnancy." She was discouraged because she had not thought she would become pregnant again. She tried to abort, without success. After the baby came, she was restless and nervous, her asthma grew worse, she was depressed and tearful, shunned social contacts, "felt nervous" and tried to run away. She remained at Glenside Hospital 30 days and seemed to respond favorably to psychotherapeutic treatment. She was not well enough to go home, but left the hospital against advice.

CASE 22. *Manic-depressive psychosis: depressive type.* A 46-year-old multipara was admitted to Glenside Hospital suffering with a manic-depressive psychosis. This was her second attack. Her case is recorded here because during her first pregnancy she was "overactive and flighty." This mental state continued until after the death of her baby, when it was 7 months old. Two months later, her mother died, and the patient went into a depressed state that lasted several months. There were two subsequent pregnancies, with delivery at term without incident. The family history was negative so far as known to the informants.

CASE 26. *Toxic-exhaustive psychosis.* A 29-year-old primipara whose previous health had been "good" had been married about a year. One sibling had been a patient at a hospital for mental diseases. Previous to delivery, physical examination revealed a loud, blowing systolic murmur, apparently of rheumatic origin. The patient developed mental symptoms soon after childbirth, and was admitted to Glenside Hospital when her baby was 2 weeks old. At first, she was fearful of insanity. At times, she had both auditory and visual hallucinations. Later, she was confused, disoriented, extremely agitated, excited, fearful, suicidal and homicidal. She developed local abscesses and ran a septic temperature for several weeks. At the end of a month, improvement began and was progressive and continuous until she went home "on visit." The duration of the psychosis was 3 months.

DISCUSSION

Emotional instability is commonly found in women during the period of pregnancy. It is not, however, necessarily caused by this state. Many of our patients, and members of their families as well, assured us that their general health during this period seemed to be better than at other times. It is highly improbable that pregnancy in itself, in a previously healthy woman, may be considered a true etiologic factor in the psychoses of pregnancy and the puerperium.

It is true that sociologic and economic factors complicate the clinical picture of pregnancy and childbirth. Inherited (constitutional) tendencies and the personality make-up of the patient are

important contributory conditions. No two persons react in the same way to the ordinary stresses of life, and individual personality trends and problems may act as predisposing factors in any mental breakdown. It is essential to study the patient as a person quite apart from her specific symptoms. Marriage calls for adaptation to a new experience. Childbearing makes more new demands on the woman, and these demands often come before she has successfully adapted herself to the personality of her husband and to the new environment confronting both of them. Maladaptation usually indicates considerable emotional conflict, which alone may produce a so-called "nervous breakdown." Feelings of guilt often result from the use of contraceptive measures, and such feelings may be of serious import when abortions have been induced or attempted, or in an extremely psychoneurotic woman.

This series of 45 cases indicates that as contributing or precipitating factors pregnancy and labor play a significant part in the psychosis occurring within three weeks after delivery and also in many cases in which the psychosis develops somewhat later. Only 2 patients in this series became psychotic as late as four months after delivery.

So far as symptomatology and prognosis are concerned, the fundamental symptoms manifested in the psychoses under consideration do not seem to differ from those of the other biogenic psychoses. Pregnancy and parturition are emotional as well as physical experiences. In an article bearing on experience as a psychiatric concept, Kahn and Cohen[12] state:

> Every individual has a *capacity to experience* which is peculiar to him. That many individuals share similar experiences is quite possible; that all individuals have the same experiences is impossible. For, each individual brings into the world *his* capacity to experience, and each individual differs from every other individual in that all his capacities, necessarily including his capacity to experience, have been derived fundamentally from the biological potentialities which he has inherited from his progenitors.

We feel justified on the basis of experience in accepting the fairly well-demonstrated postulate that individual biogenic weakness, although the resistance to environmental conditions may be only temporarily lowered, underlies all the biogenic psychoses. This study does not show that toxic-exhaustive factors, which are apparently the result of pregnancy and childbirth, produce symptoms that are different from those accompanying other toxic-exhaustive psychoses. Psychologic stresses, and the varying reactions of the patient to them, are of the same relative importance here as in the other biogenic and psychogenic psychoses. In many other types of mental disorder occurring in adult life, one may find a history of long-continued economic stress given as a contributing cause. And unquestionably this type of strain in itself produces a profound anxiety state in the woman who is about to bring a child into the world. Other situational factors — such as incompatibility between husband and wife, an undesired pregnancy, a definitely unwanted child, a feeling of inadequacy about assuming the responsibilities of motherhood, and a previously sheltered life without having achieved emancipation from her own protective mother or father — are noted as contributing factors. One or more of these conditions recurred often in the 45 cases here considered.

Conclusions

Mental disorders associated with pregnancy and the puerperium as reported by many observers may easily fall under the usual familiar nosologic headings. Many observers have found nothing specific in psychoses occurring post partum. However, as contributing and often as precipitating factors, these two incidents in a woman's life should not be disregarded.

The term "puerperal insanity" will doubtless fall into disuse, and a better term will be substituted. At present, it seems to be commonly used only in a descriptive sense.

Owing to emotional and functional alterations during pregnancy and the puerperium, many women are, by reason of poor heredity, unable to go through the long period of gestation without a break in mental and emotional integrity during the puerperium.

Endocrine and metabolic imbalances may play a large part in these and other psychoses. Evidence of their etiologic significance is insufficient for any more definite statement to be recorded.

The great need for early educational preparation for parenthood for both sexes cannot be too often emphasized, or too often stated in nonmedical as well as medical literature.

Prenatal care, including, when indicated, psychiatric study and psychotherapy of a re-educational character, should be readily available to every pregnant woman, no matter what her social and economic status.

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PRESIDENTIAL ADDRESS*

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CONCORD, NEW HAMPSHIRE

I VIEW my task this evening as definite and inescapable. I must give a presidential address, saying words and expressing thoughts befitting a time-honored anniversary, yet finding myself without the wisdom and special ability the occasion calls for. Not of the same mold as most of you, with an experience and training of quite another sort, I have from my medical cradle been nurtured in a cloistered life that scarcely fits me for a real understanding of the things that are of interest and concern to you in the private practice of medicine, and I frankly confess to being considerably confused and uncertain of the form and substance this address should take.

Had I followed the example of my illustrious predecessor (and, whom better could one pattern after?), I would, without more ado, have prepared a paper for the afternoon session. But since a president's paper, being ex cathedra, is not open to discussion or even question as to its validity or scientific integrity, I hesitated. Ez Jones, of course, would have no need for any such hesitation.

Therefore, it has seemed best to say whatever I have to say, here at this banquet where the warmth and encouragement in these many feminine eyes will serve to dilute and weaken what otherwise would have been just the concentrated essence of a cold stare from a roomful of scientifically critical confreres.

Had I elected to read a paper this afternoon, being mindful of the old admonition that "the shoemaker should stick to his last," I believe I would have said something about that very much misunderstood and therefore quite unhappy branch of medicine known as psychiatry. And, mark you, at the very onset I would have boldly insisted that psychiatry must now be recognized as an important aspect of general medicine, as well as a weird specialty, in spite of the woman who said to one of my kind, "If you were only a physician instead of a psychiatrist I should like you to take my husband's blood pressure," and in spite of the man who commiseratively said to me, "Now if you were only one of them M.D.'s you could probably get some new tires."

Psychiatry is an aspect of general medicine because mental and emotional factors play such a significant role in invalidism. More and more are we being oriented to the concept that there is a definite relation between physical and mental disorders. More and more is the value of treating the total personality, the patient as well as his disease, being better appreciated.

Thus, the true function of medicine must be the study and treatment of man, himself, as well as his organs and cells, if the practice of medicine is to be kept very much above the level of a glorified veterinary science, and I dare say even a horse doctor would admit that if he could only tell what was on the horse's mind he could the better treat the horse's belly.

From the practical standpoint of treatment, however, it is still quite useful to consider the individual not as one person but as two, — a physical man and a mental man, — the treatment required varying according to which man it is, provided, of course, we bear in mind that it may be both.

Therefore, in our conquest of disease we should, to use a modern idea, wage a total war. Yet how can physicians wage total war when lawyers get so much in the way? How can we wage total war against pathologic drunkenness, for example, when the legal dictum is that a man may drink himself into the grave and his family into destitution unless he can be certified as "insane," generally a difficult thing to do because incompetency, the legal test, is rarely manifest? And how can we wage total war against mental disease in general when the law hinders early treatment by making certification necessary for admission to a remedial hospital, branding the mild or recent case with the word "insane," with all the unfortunate consequences that flow therefrom? Lawyers are naturally concerned with arranging for the protection

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of the public, but physicians chiefly interested in the health of the public ask only how the sufferers can best be treated to bring about a cure.

In the previous war, thousands of soldiers were treated for mental breakdown, without being certified as "insane." Is there any difference between a soldier who loses his mind and a mother who develops mental symptoms while producing her country's defenders?

All this is just the fringe of a large subject, and my thoughts about it are some of the things I might have put down in a medical paper to be read before the learned doctors in scientific session; however, they hardly do for a presidential address and I shall pursue them no further now. I know you are beginning to say to yourselves, "If psychiatry is all he seems to think it is, then why is the sick Hitler still at large?" That Hitler is mentally sick, we psychiatrists well know, and we are closely following his pathologic performances, including his attempts to destroy the mental health of others based on his "war of nerves" and his verbal distortions of truth. Like many other mentally ill persons he makes promises today which are broken tomorrow. Like others mentally ill, he rationalizes all his actions by verbal distortions.

What obvious psychopathy there is in the Hitler idea of German superiority — the master race in a world of slaves! What claustrophobic air hunger in his constant bleating for more and more *Lebensraum!* What homicidal mania in his ruthless obsession to destroy nation after nation! What megalomanic overestimation of his strength in his ill-judged attack on Russia! The clever, the brilliant Hitler — never was there greater imposture.

Then, why is he still at large? In that circumstance lies the most fateful diagnostic failure in all history. As far back as 1923, when this pseudo-Caesar, who could not even pass his school examinations and was really never a good paper hanger, started his first uprising, or *Putsch*, against the existing German government, he was arrested, put under observation as to his mental condition, and Oswald Bumke, professor of psychiatry at the University of Munich, just plainly muffed it. Instead of recognizing that he had on his hands a dangerous paranoiac who should be locked up indefinitely, he merely called the sick Hitler a fanatical psychopath and all he got was six months in jail. Now that is the story of why Hitler is still at large, a story that, so far as I know, has never been published and which I would prefer not to have go beyond this room because of the medical ethics involved as I would not wish to injure Dr. Bumke's medical reputation.

Having revealed the story of this egregious psychiatric blunder, I may as well come thoroughly clean and admit that for centuries psychiatry was the most backward and, therefore, the most belittled of all medical sciences. The Renaissance that terminated the dark ages for medicine in general did not apply to psychotherapy, and for a long time this field was renounced by medicine itself and left to religion and charlatanism. With the exception of healing through religious faith, treatment of the mind came to have a bad name on account of its association with ignorance, superstition and fraud. In other words, mental medicine, like some people, has an unsavory past, which it has not been easy to live down. However, the presence of some form of mental treatment has always been inevitable on account of the nature and needs of psychic difficulties, and if the impostor and quack have flourished in this field it is largely because official medicine has refused to recognize psychotherapy as having a legitimate place in medicine. But, after all, even if psychiatry was begotten in charlatanism, the surgeons are not looking us down very hard, because everybody knows they descended from the barbers. (Perhaps the surgeons will feel better if I say "ascended" instead of "descended.")

Well anyway the first approach to anything like a scientific psychotherapy is associated with the name of Mesmer, an eighteenth-century physician of sound culture who first grasped the principle of so-called "suggestive psychotherapy" in the hypnotic state. Unfortunately, he encumbered his art with fantastic theories of animal magnetism (now taken over by the Christian Scientists) and some strange fluid that passed from operator to patient, and gave his treatments in a setting of oriental mystery with the aid of magnets, wires and other paraphernalia. Mesmer was repudiated. Nevertheless, he is immortalized in the word "mesmerism," and some of his work stripped of false theories and sideshow trappings must be accepted as the basis of modern psychopathology and the first substantial contribution to a sane psychotherapy.

Later, an English surgeon turned his attentions to the principles of Mesmer and, with the clear vision of a scientist, soon saw that he was not dealing with strange body fluids or animal magnetism but with purely mental phenomena. Then followed Charcot, who forced a reluctant medical world to accept the fact that a wide variety of illnesses can be produced by purely mental causes and should be treated by mental means, and then Janet, who was the first to recognize that nervous illness is not something that seizes on the patient suddenly but is instead the end product of the whole life history and psychologic evolution.

Finally, we had Freud with his theory of the unconscious mind and his technic of psychoanalysis,

around which there still rages much controversy but whatever one's opinion about the Freudian theories and technic, it must now be conceded that from being static, psychiatry has become dynamic, from being chiefly descriptive, it has now become interpretive, and from treatment en masse, it is now recognized that each patient is an exquisitely individual problem.

In this war that engulfs us, there is more need than ever before for psychiatry's contribution. Those mentally unfitted for army service must be eliminated. Civilian morale must be maintained. So loosely, so ambiguously, has the term "morale" been used that it has almost lost its meaning. Real morale is built neither by slogans, catch phrases, song and dance acts nor even by movie actresses on whirlwind visits through army camps. At best, this is only diversion and has little to do with basic principles. If you would know morale, observe the Russian people and the Russian army. Obviously, they are a people who believe very deeply in the things for which they are fighting. It is their faith in themselves and their institutions that is sustaining them. There are no slogans, dances or even hostesses in the Russian camps. May we too learn how to sustain ourselves through the weariness of continuing frustration! There is work, sacrifice and responsibility for everyone. Not a vestige of the world we have dimly anticipated for those who follow will continue if we fail each other.

I spoke in the beginning of my feelings of inadequacy for meeting the challenge of this occasion, but it is clear to me now that it should not be difficult to pay tribute to the strength and harmony of this venerable society.

One of my duties this year was to send a letter of felicitation to the Connecticut Medical Society on the celebration of its sesquicentennial. I wrote:

> The New Hampshire Medical Society congratulates the Connecticut State Medical Society on the approaching one hundred and fiftieth anniversary of its founding. We recall the pleasurable satisfaction we experienced at our sesquicentennial way back in 1941. Connecticut did well to emulate New Hampshire a century and a half ago in forming a state medical society and in profiting from the longer experience of the older brother in the right way to maintain the vitality necessary for a long life. If you again emulate New Hampshire in the form of celebration you deem worthy of such a notable event, you will make it a festival such as will give your founding fathers at least one very restless night.

I tell you this because in the first place you are entitled to know what kind of rascally advice your president gave to our neighbor, and secondly because it is pleasant to recall and relive for the moment the very happy celebration of a year ago. To be sure, it was quite a let-down for an august and dignified society such as ours, but very helpful and mentally healthful, if indulged in only once in a hundred years, more or less.

One hundred and fifty years is indeed a long life, and there must have been some need in the hearts and minds of the members of this society to explain its prolonged virility through many social and economic stresses.

The most valuable purpose of such an organization as ours is the improvement of its members by advance in knowledge. No class stands in greater need of getting together frequently. You are called on to decide complicated problems involving the well-being, the very lives, of your fellows. The experience and knowledge of even the most learned is limited, and it is easy to become rusty. There is no corrective equal to discussion with others.

To be sure, much of what has been said here during the past two days will be published in the *Transactions*, but the man deceives himself who fancies he can derive the same benefit from reading the transactions as he would from attending the meetings, for he misses the second great object to be obtained in a society like this — the binding of its members together by friendly association and personal acquaintance. More than ever, in these dark and tragic days, do we need the trust and friendship of each other.

I still view with astonishment my election to this presidency. I was deeply touched by the honor and shall always think of it as an index of your democracy.

However, beyond this one election, I do not see that your habits are much akin to those of our great American democracy, for although fate has made me a war president, it does not escape my observation that you have no apprehension about "swapping horses while crossing a stream." I salute the new horse and assure him the load is not heavy. There are draft horses to do the work.

Finally, in surrendering the gavel, I can at the moment think of only one thing nicer than being your president and that is being welcomed into that charming coterie of aging men — your ex-presidents.

THE NEUROLOGIC ASPECTS OF DEFECTS IN SPEECH AND READING

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MUCH of man's success depends on the use of words. There is surprisingly little interest on the part of the medical profession in how people use words. Some time ago, I thought it would be of value to establish a clinic to study patients and their use of language. "Language" has many connotations, and perhaps a better term would be "wording," which means how words are produced in spoken and written form, and how they are received by ear and eye. This study has been the function of the Language Clinic at the Massachusetts General Hospital.

Of the patients who come to the clinic for study, the youngest group and one of the largest groups is that of children of three and a half, four and five years of age who do not talk or do not talk in an understandable way. These children usually appear two or three weeks before the opening of school, when worried mothers bring them in saying, "Johnnie doesn't talk yet, and he'll have to learn before Labor Day when he goes to school." Or else the worried parent who has waited still longer brings the child in during October with the sad tale that the child has been rejected from school because he could not talk or because his speech was not understandable. Sometimes, parents are worried earlier, and the children are seen before they are ready for school. Such parents are usually apprehensive about the child's intelligence, for intelligence and ability in wording are synonymous to many. This, of course, is not true.

In the study of this group of poor talkers, the late talkers and poor talkers can be divided into several groups. In the first place, there are some who do not talk apparently because of poor intelligence. Such a patient, a little boy of seven, was brought to the hospital recently. He had for the second time been rejected from the first grade because his speech could not be understood and also because he could not fit into the group situation at school. The teacher thought that his lack of development in talking made it impossible for him to fit in with the other children. So far as could be ascertained, the child had no physical defects as judged by an examination of his sensory-motor system and his general health. He had had but little illness. His speech was quite difficult to understand. He used many words that could be understood only by his mother, who was adept at interpreting them. A careful psychometric examination was made, nonlinguistic tests being used, and the child's mental age was found to be far below his chronologic age; in fact, it was only a little above three years of age. In such a case, then, in the absence of other causative factors, it is fair to say that poor intelligence, as measured by suitable intelligence tests, is the cause of poor wording. This is not always so, for children with very low mental ages sometimes manage to talk quite well.

Another subgroup of late and poor talkers includes children with deficient hearing. These children are puzzling, for they hear some sounds, and hence the fact that they are partially deaf escapes many observers, including their parents. They may like to listen to the radio, and they may jump when a dog barks or a bell rings, so that obviously they are not deaf. As a matter of fact, they often respond to commands, and the mother quite rightly says that they understand what is said and, therefore, they must have hearing. Careful evaluation of these cases, however, may show that a good deal of hearing has been lost. Some of these children have a loss of hearing in the high-frequency range. This is a selective type of deafness, excluding many of the consonant sounds, such as *s*, *sh*, *t*, *th*, *f*, *v* and a few others, from the child's hearing. Such children are unable to reproduce the speech that they do not hear. Their speech is quite unintelligible, for it is without so many of the sounds one is accustomed to hear. Some of these patients have a selective or high-frequency type of deafness; others are slightly deaf in all ranges of frequency. There are a good many such cases; certainly, there are enough to make one realize the advantage of having a painstaking evaluation of a child's hearing done by a competent otologist before any other therapy is begun. Hearing aids in these cases may be of great assistance, and other measures may improve the child's speech.

There is a third subgroup of nontalking children. This, in my experience, is a small one. It consists of children who seem to hear sounds but do not associate meaning with words. They have been described in the literature as "word deaf." Such children obviously do not respond to their parents' words, and are usually very much interested in movements, expression of faces and all the other meaningful signs they can observe.

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Most children, however, who do not talk well or do not talk at all at the expected ages seem to have no physical or mental defects; that is, their intelligence is average or better, their hearing is adequate, they show no physical defect of the organs of speech, and there is no evidence of brain or nervous-system disease. Yet they talk late, or, when they talk, use poor speech, which may be described as very marked baby talk. This group, which is large and in which there is no demonstrable defect, is a particularly challenging one, which I wish to call "motor speech delay."

Second to these children who do not talk well at the proper time come the group who talk with a stutter. I use the terms "stuttering" and "stammering" interchangeably, for there is no difference in the underlying processes. Some persons have difficulty because of repeating one sound over and over again; others, who cannot get a single sound out, have what is known as a "block type" of stutter. These disturbances may be so severe as to be tremendous handicaps, or they may be so slight as to escape observation. The hemming and hawing of many speakers is a form of this same thing. One usually sees little children about to start school, or who have just started school, who stutter. Their parents quite rightly feel that the handicap may be disturbing to school progress, and hence the patients are brought in for help. Frequently, the stutterer is an adolescent whose career in school has been a difficult one because of his speech defect. At adolescence, he wishes to develop a more useful personality and, to this end, to eradicate his speech defect. Stutterers who present themselves for help consequently range in age from early childhood through the growing years. One can find among them only one common denominator — the fact that they stutter. Their organs of speech and neurologic control vary as among other persons; there is no commonly found physical defect to explain the stuttering. Stutterers, then, are a group gathered together because of their symptom.

There are, of course, large numbers of children who come in to the Language Clinic because of organic defects interfering with speaking. In enumerating such defects, faulty alignment of the teeth should be mentioned first. I hasten to add that it is surprising how well children can talk with very poorly aligned teeth, and it is only rarely that the teeth are the cause of poor speech. Cleft palate is another organic defect conditioning speech. The operative repair of cleft palate is now so well performed that it is a pity that more patients do not talk better after the operation. That they do not, in my opinion, is because of inadequate postoperative training. If a child is given an altered equipment for the production of speech, he must be taught properly how to use it, for he still has in his memory, as correct speech, the sounds so poorly enunciated with his cleft palate. As the operative age becomes lower and lower, this hazard becomes less and less, but even so, the soft palate needs to be stretched and used properly if good speech is to result. Among organic defects influencing speech, deafness should be referred to again. Suffice it to say that many children apparently deaf since very early childhood escape notice until they reach the upper grades in school. Finally, there is the large group of organic defects whose lesion is in the nervous system as a result of cerebral birth injury. Many of these children have poorly co-ordinated movements or spastic movements, all of which are reflected in their speaking. In some of these cases, particularly when there seems to be good intelligence, some improvement in talking can be gained with good speech training.

An interesting group in the clinic might be classified as suffering from misuse of the voice. These are children whose speech is defective because, along the line of their development, they misused their voices for one reason or another. In little children, the misuse sometimes follows tonsillectomy when, owing to a sore throat, the child splints his soft palate for a prolonged period, and then develops the habit of talking with a good deal of hypernasality. His speech sounds almost as though he had a cleft palate, and the defect dates from the time of the tonsillectomy. This defect is the child's response to a painful throat, and can readily be overcome soon after the operation by proper throat exercises before the restriction of voice becomes fixed. At puberty, not infrequently, boys misuse their voices and report with a speech disturbance as a result. An example of such a patient was a boy of thirteen, rather small for his age, whose mother brought him in because of his high squeaky voice, which could hardly be heard. He had developed this voice during the six months preceding his hospitalization. On examination, it was found, when he was asked to make certain sounds, that he had a very good, unusually deep bass voice. When questioned about this, the patient rather shamefacedly admitted that he did have this sort of voice but he was embarrassed by it because it seemed so deep and large for a small fellow. He had developed a falsetto speech to save himself embarrassment. Another somewhat similar case is that of a young man of thirty-five who came in with an excessively husky voice. He had been carefully examined in the Throat Clinic, and no abnormality to which the voice could be ascribed had been found. On examination, it was found that this patient had sung in a boys' choir. He had had a very fine soprano voice. During pu-

berty, he continued his work in the choir and found that with some effort he could continue the soprano parts with which he had been so successful. Some time later, he became a singer with a jazz orchestra and was popular because of his lovely, high, crooning voice. It was after using his voice in this way for quite some time that he found it increasingly difficult to speak above a husky whisper. When he was seen in the clinic, he could still raise his voice to a high soprano falsetto tone for singing, but his voice for speaking seemed to have suffered as a consequence.

Another large group of patients seen in the Language Clinic are those who have lost the ability to use language. These are the cases of aphasia not infrequently seen in hospital and general medical practice. Aphasia is by no means limited to aged people who have had shocks, associated with hemiplegia. A good many of these patients are young people who have had vascular accidents or other trauma to the brain and who apparently have years of life ahead of them. A study of aphasia helps one to gain an understanding of the language processes. The patients who lose ability in wording may have a defect chiefly in their ability to say words. Another group of aphasic patients show their greatest defect in ability to read print. They become relatively alexinic. Finally, a third group show their greatest defect in ability to understand the spoken word. I do not believe that any one of these parts of the language processes can be affected without causing a defect in the other parts as well. However, the proportion of one defect in relation to the others is a major factor. By evaluating this factor carefully, one learns much that is of great neurologic value regarding the cerebral localization of the lesion causing the aphasia. One also learns that there is an interrelation between the different areas of the brain involved in the language processes. Thus, when the lesion is in the motor speech area in the frontal lobe, talking may be completely lost, whereas understanding of words and of print is only partially lost. This interrelation is of particular consequence from the therapeutic point of view. In planning retraining exercises for the patient who is aphasic, one can do much by working through the spheres of language relatively intact, to improve the portion of language most affected. A recognition of this fact also points the way for help in other language problems without recognizable abnormality. Thus, one can remedy the speech defect by working through the auditory and visual speech areas, that is, through the hearing of words and the reading of print. Similarly, the reading disability is remediable by the linking of poor performance in this one language process with better performance with the spoken word.

With these facts in mind, it may be readily recognized that no satisfactory work with disorders in wording can be carried on without a thorough study of aphasia. In the sphere of language, as elsewhere in the study of man's biologic processes, knowledge of function is increased by the observation of changes associated with disease.

I have described, then, various groups of patients whose chief difficulty is their output of words. Another large group are examined because of difficulty in school. These children may be regarded as having a learning difficulty. It is self-evident that a child's learning in school is by means of his wording. Facts and school experiences come through his ears as the spoken word, and through his eyes as the printed word. He expresses his thoughts and thereby gains experience, his knowledge being measured by his teachers through more wording, his speech and his written productions. Since wording is the medium by which learning in school is accomplished, learning difficulties frequently resolve themselves into difficulties in wording. The implications of this fact are tremendous and are generally overlooked.

In considering a child's learning difficulty, one of course investigates his general health, which may be a retarding factor, and his physical equipment, particularly his sense organs and his general motor equipment. Further investigation leads to attempts to evaluate the child's intelligence. There are numerous forms of psychometric examinations, all of which evaluate intelligence merely in terms of the given test. Many of these tests are given through the medium of wording, and since many of the children investigated are deficient in this respect, the limitation in language rather than one in intelligence is frequently measured. I know of no test that is foolproof in this respect. Of course, the most unreliable for this purpose are group intelligence tests, which require successful use of language before the test can be mastered. Individual intelligence assays, then, must be employed. Even these lean quite heavily on oral directions, and some of them on printed material. Moreover, since they presuppose considerable experience in language, many of these patients, who are deficient in language skill, are at a disadvantage. The most helpful means of evaluating intelligence for this purpose has been to use a fairly large battery of tests, some of which depend almost entirely on performance. When this is done, one usually gains a reliable idea of the child's academic potentialities from the point of view of his intelligence.

One inevitably sees children whose defect is discernible by means of a careful psychometric examination. Such children are described as having a somewhat retarded intelligence, and in some cases this seems to be the explanation of their performance. There are many more, however, who in terms of mental tests have adequate intelligence and whose scholastic achievement is far below what might be expected of them, in view of their chronologic and mental age, and even in view of their grade placement. The so-called, mythical "average child" of eleven years having an average intelligence quotient of 100 should have a mental age corresponding to his chronologic age, and on the basis of average public-school performance, he should be entering the fifth grade in school; theoretically, his performance in arithmetic, reading and spelling, to mention only the fundamental subjects, should be the average for children beginning fifth grade. Of course, such an average child does not exist, and one finds wide variations from this norm. Thus, I have already described the child whose variation seems to be one of retardation in the mental age, accounting for retardation in scholastic achievement and possibly in grade placement. However, some of these children have a mental age that is average or much better than average for their chronologic age, and their performance in school lags well behind what would be expected of their mental age. This inconsistency must be explained. In the absence of physical and mental defects, one must look further for an explanation. When such a child's academic achievement is measured, his performance in arithmetic is generally far superior to his performance in reading and in spelling. When the use of words, printed and written, is concerned, the child's greatest failure is apparent. Here, then, are retarded children whose problem is not so much learning per se as it is the difficulty in wording, particularly in reading, writing and spelling. These cases have been described by others as congenital reading disabilities.

* * *

In this study of cases seen in a clinic devoted to language problems, the difficulty of some patients is explainable on the grounds of organic defect. Others show no organic defects, and yet their wording is defective. Among them, I have described those who acquire the ability to talk late and who talk poorly; one recognizes little children of three, four and five having a motor speech delay. Then, there are the large group of stutterers, ranging from the very severe stutterers to those whose residual defect is only a slight hesitation in speech. On the auditory side, there are those who do not seem to understand words and hence have difficulty in reproducing them. These are cases of central-type or word deafness. Finally, there are the large group of reading disabilities.

These patients have a common denominator — their heredity. One finds, when investigating the heredity of any one of these types of cases showing difficulty in wording, that in the family there are cases of right-brainedness as well as the usual left-brainedness. By this, I mean a dominant right cerebral hemisphere. The simplest measure, although not always an accurate one, of hemispheric dominance, is the handedness. Thus, a person having right-cerebral-hemisphere dominance is usually left-handed. The dominance for handedness and for language performance is usually, although not always, in the same hemisphere. Of course, right-brainedness does not always occur in a person whose performance in language is defective, but it does occur in his family. Moreover, when one type of hereditary language defect occurs in a family, other types are likely to appear in members of the same family. Thus, in the family of the stutterer, there are cases of late development of speech, as well as reading disabilities and so on. It seems then, that one is dealing with a hereditary syndrome. A possible explanation of this fact lies in what has been learned of the language function by a study of aphasia: the language function is regulated by the dominant hemisphere, the opposite hemisphere having little, if anything, to do with wording. The patients concerned have a combination of opposing dominance. They are those who have inherited a tendency toward a dominant right as well as a dominant left hemisphere. This inheritance sets the stage for poor language performance in any of the respects already described. These children with a poor hereditary setup for language can improve their performance with careful training. To improve performance, it is well to recognize the fundamental lack of ability, for unless one does, methods planned for improvement will miss their point. Furthermore, unless one understands the interrelation between different parts of the language process, — talking, reading and hearing words, — one misses a chief avenue of therapy in the field of language problems.

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SULFATHIAZOLE OINTMENT IN THE TREATMENT OF PYOGENIC DERMATOSES*

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SINCE the advent of sulfanilamide and its derivatives, a beneficial clinical result in many dermatologic diseases has been effected. Beinhauer, Knoll and Perrin[1] observed the response to sulfathiazole, given orally, in 103 patients with sixteen dermatologic diseases. During the course of the oral administration of the drug, many toxic symptoms may arise, such as headache, nausea, fever, burning and itching of the eyes, injection of the scleras and conjunctivas, dermatitis and exquisitely tender and swollen joints, with effusion, leukopenia, hematuria and anuria.[2] Moreover, Spink and Paine[3] treated 16 patients with localized staphylococcal lesions, in 15 of whom the organisms were found in the blood stream. After sterilization of the blood stream by oral sulfathiazole, organisms could still be cultured from the localized lesions. Thus, it seems unwise to subject patients with minor pyogenic dermatoses to the oral administration of the drug, since the toxic manifestations may be severer than the disease and the potential danger of invasion of the blood stream from localized lesions exists even after the blood has been sterilized.

A much simpler method in treating the pyogenic dermatoses is indicated. MacKenna[4] observed that when crushed M and B 693 was dusted on cases of impetigo and ecthyma and covered with zinc paste and a light dressing, the time required to cure the average case was reduced. The same observation with sulfathiazole has been made at the Boston City Hospital since November, 1940. A crude ointment was made by trituration of sulfathiazole tablets and mixing of the powder in a base containing equal parts of lanolin and boric ointment. A more refined ointment was then obtained‡ in which the sulfathiazole was in a suspended state and the size of the particles in suspension approached that of a colloidal one.

Forty-two patients were treated with the crude and refined ointments, with satisfactory results. Twenty had impetigo, 7 pyoderma, 7 eczematoid dermatitis, 1 a recurrent herpes simplex with secondary infection, 1 a herpes zoster with secondary infection, 3 infantile eczema with secondary infections, 2 disseminated neurodermatitis with secondary infection, and 1 a stasis eczema with secondary infection. Cultures were obtained in 11 of these cases, and the predominant organism was found to be a hemolytic *Staphylococcus aureus* (Table 1).

The patients were instructed to cleanse the involved areas once daily with soap and water, to remove all crusts, and to apply the ointment every two hours during the day and once before retiring. The ointment was applied frequently, since Fleming[5] observed that bacteria and peptones inhibit the bacteriostatic action of sulfanilamide and since peptones are produced by the action of the proteolytic ferments of disintegrating leukocytes on proteins.

TABLE 1. *Results of Bacteriologic Studies.*

PATIENT	DISEASE	ORGANISM FOUND ON CULTURE
D. C.	Impetigo	Hemolytic *Staph. aureus*
W. C.	Impetigo	Hemolytic *Staph. aureus*
B. D.	Impetigo	Hemolytic *Staph. aureus*
J. M.	Impetigo	Hemolytic *Staph. aureus*
I. E.	Pyoderma	*Staph. albus*; a few colonies of *Staph. aureus*.
S. E.	Pyoderma	*Staph. albus*, some hemolytic
A. L.	Pyoderma	Hemolytic *Staph. aureus*; a few colonies of hemolytic streptococci.
T. R.	Eczematoid dermatitis	Hemolytic *Staph. aureus* and streptococci
N. D.	Eczematoid dermatitis	Hemolytic and nonhemolytic *Staph. aureus*
T. D.	Infantile eczema, with secondary infection	Hemolytic *Staph. aureus*
P. C.	Stasis eczema, with secondary infection	Nonhemolytic *Staph. aureus*

TABLE 2. *Response of Lesions to Sulfathiazole Ointment.*

DISEASE	NO. OF CASES	AVERAGE HEALING TIME
		days
Impetigo	20	4.6
Pyoderma	7	3.7
Eczematoid dermatitis	7	6.7
Recurrent herpes simplex with secondary infection	1	4.0
Herpes zoster with secondary infection	1	9.0
Infantile eczema with secondary infection	3	6.0
Disseminated neurodermatitis with secondary infection	2	3.0
Stasis eczema with secondary infection	1	4.0
Average		5.2

The length of time for the pyogenic lesions to respond was from three to nine days, the average being five days (Table 2). The strength of the

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‡The refined ointment was supplied through the kindness of Abbott Laboratories, North Chicago. It consisted of a 5, 10 and 15 per cent suspension of pure sulfathiazole powder in an ointment preparation containing diethanolamine oleate, mineral oil (premier, white), petrolatum alba (*U.S.P.*), beeswax (*U.S.P.*) and distilled water.

ointment, whether it was 5, 10 or 15 per cent, made very little difference in the rate of rapidity with which the lesion tended to heal. When patients with impetigo who were treated with sulfathiazole ointment were compared with those treated with other medications, the results were equivalent or more satisfactory (Table 3).

TABLE 3. *Comparison of Results in Impetigo.*

PREPARATION	NO. OF CASES	AVERAGE HEALING TIME
		days
Sulfathiazole ointment (5, 10 and 15 per cent)	20	4.6
Ammoniated mercury ointment (2 per cent)[6]	25	5.0
Compound chlorhydroxyquinoline ointment[6]	25	4.7
Metaphen (1:500) in flexible collodion[7]	234	8.0
Ammoniated mercury and colloidal kaolin lotion[8]	79	15.0
Ammoniated mercury (2 per cent) and yellow oxide of mercury (0.5 per cent) ointment[8]	32	26.9

The following are typical case reports:

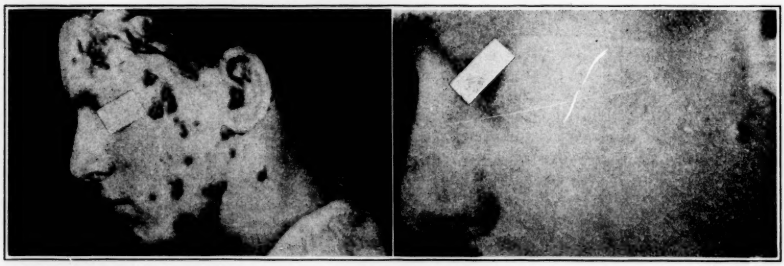
CASE REPORTS

CASE 1. W. S., a 17-year-old boy, was admitted to the Boston City Hospital in January, 1941, with impetigo of the face and chin of 1 week's duration. No results were obtained from the use of various local medications. Examination revealed multiple, heavily crusted, oozing lesions on the left side of the face and chin, which were covered by a yellowish medicament. The patient was instructed to remove the crusts with soap and water and to apply a 5 per cent sulfathiazole ointment (crude form) to the lesions every 2 hours during the day. The patient responded, and was discharged in 4 days with all the lesions healed and with very little evidence on his face of where they had been present (Fig. 1).

CASE 2. T. L., a 22-year-old woman, entered the Out-Patient Department of the Boston City Hospital in March, 1941, with an infectious eczematoid dermatitis involving the pinna and external auditory canal of the left ear of several weeks' duration. Examination showed an oozing, crusting dermatitis, with moderate edema. The patient was instructed to wash the lesions once daily with soap and water and to apply a 10 per cent sulfathiazole ointment (refined form) to the lesions every 2 hours during the day. In 4 days, the superficial infection, as evidenced by crusting and oozing, had disappeared, and healing took place in 8 days (Fig. 2).

CASE 3. P. C., a 60-year-old man, was admitted to the Boston City Hospital in April, 1941, with a stasis eczema and secondary infection of the left leg of several months' duration. Examination revealed moderate edema of the left leg, with marked varicosities and superimposed secondary infection, as evidenced by oozing and crusting. The patient was placed on bed rest and a daily regime of cleansing with soap and water and application of a 5 per cent sulfathiazole ointment (refined form) to the lesions every 2 hours during the day. The superficial infection responded in 4 days, and healing took place in 10 days.

In the 42 patients treated with the crude and refined forms of the sulfathiazole ointment, no local toxic effects were observed. To determine whether absorption of the sulfathiazole took place into the blood stream, 5 gm. of sulfathiazole, in ointment form, was placed on the arms of 4 patients, 2 with normal skin and 2 with generalized erythroderma and secondary infection. The preparation was allowed to remain in place for forty-eight hours by the aid of dressings. During this period, samples of blood were collected at the end of six, nine, twelve, twenty-four and forty-eight hours. At the times when blood was collected, all urine was saved. Sulfathiazole levels were determined on both the blood and urine specimens, and it was found that minute amounts of the drug were absorbed into the blood stream and excreted into the urine. Thus, it was concluded that sulfathiazole in ointment form is less likely to produce the systemic toxic manifestations that one may observe during the administration of the drug orally.



A B

FIGURE 1.

A *shows impetigo of the face on admission to the hospital;* B *shows the same area after four days' use of 5 per cent sulfathiazole ointment (crude form).*

Summary and Conclusions

Forty-two patients with eight different dermatologic diseases, which were caused by pyogenic organisms (mostly hemolytic *Staphylococcus aureus*) per se or were secondarily infected, were treated with an ointment containing sulfathiazole.

The strength of the sulfathiazole made little difference, the infections cleared more quickly, as a rule, than infections treated by other means, no toxic effects were observed, and the application of sulfathiazole to the skin for forty-eight hours caused only insignificant amounts of the drug in the blood stream.

From the results obtained by this method, it is believed that the use of the sulfonamides, orally, in the treatment of minor pyogenic dermatoses is not warranted because of the danger that toxic manifestations, which can be more serious than the dermatosis itself, may develop, because invasion of the blood stream from localized lesions can occur after discontinuance of the drug orally, and because the use of the drug should be reserved for far more serious illnesses.

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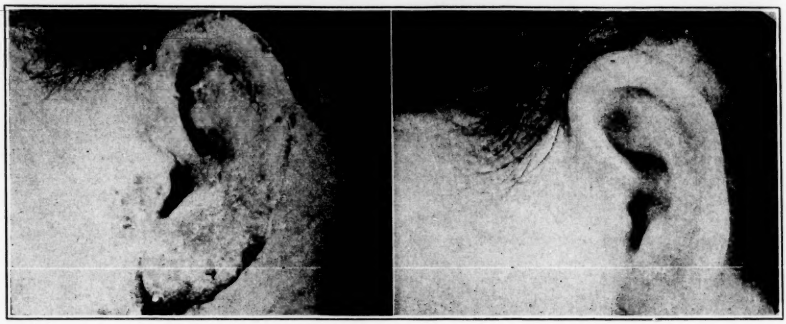

A B

Figure 2.

A shows an impetiginous eczematoid dermatitis involving the pinna and external auditory canal; B shows the same area after four days' treatment with 10 per cent sulfathiazole ointment (refined form).

HEMATEMESIS DUE TO RUPTURE OF AN AORTIC ANEURYSM

Report of a Case

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THE history of aneurysm is very old. Galen[1] knew external aneurysm well, and in the second century, Antyllos[1] devised his operation of incising and emptying such a sac, inclosed between ligatures. Internal aneurysm was recognized first by Fernelius[1] in the sixteenth century. Later in the same century, Paré[2] suggested the relation of aneurysm to syphilis. Lancise,[3] in 1728, classified aneurysms, but not in the modern sense. Hunter,[3] in 1757, used the terms "true" and "false" in the modern sense and also described the symptoms of pressure he encountered in 5 cases of aneurysm. Corvisart,[4] in 1812, described various signs and symptoms; he called attention to the thrill, retromanubrial dullness on percussion and inequality of the pulses. Oliver,[5] in 1878, described the tracheal tug. Thoma,[6] in 1888, wrote concerning the pathology of aneurysms. Döhle,[7] in 1895, discussed the microscopic changes in syphilitic aortitis, and finally Reuter,[8] in 1906, described the *Treponema pallidum* in the wall of the aorta in aortitis, and by so doing established the importance of syphilis as a cause of aneurysm.

The disease occurs more frequently in males than in females, in a ratio of 5:1. It is five times more frequent in Negroes than in Whites. Although aneurysm occurs at all ages, the greatest incidence is between thirty-five and fifty-five. Boyd,[9] in an analysis of 4000 cases of aortic aneurysm, states that post-mortem statistics indicate that aneurysms account for 0.1 to 0.5 per cent of deaths in American cities. According to Kampmeier,[10] the incidence of this condition has decreased by more than half in the past decade.

According to Boyd, the ratio of aneurysm in various portions of the aorta is approximately 10:7:3:1 for the ascending, transverse and descending segments of the aortic arch and the descending thoracic aorta, respectively.

In practically all cases, syphilis is the cause. Arteriosclerosis, tuberculosis, trauma and mycotic infections play a very minor role.

The diagnosis is based on signs and symptoms and x-ray findings. The physical signs of thoracic aneurysm are too well known to require enumeration here. It may be comparatively easy to recognize a classic case of aneurysm after a careful physical examination. However, in many cases, as in an aneurysm of the upper descending aorta, the process may be so latent and the signs so negligible that recognition may tax the ability of the most experienced clinician. In such cases, the diagnosis is made on suspicion or incidentally at x-ray examination. Osler[11] wrote, "There is no disease more conducive to clinical humility than aneurysm of the aorta."

The following case of aneurysm of the descending segment of the aortic arch is reported because the condition was undiagnosed and because the patient died of an unusual complication — rupture into the esophagus.

CASE REPORT

A 58-year-old clerk entered St. Luke's Hospital because of hematemesis. For the previous 2 or 3 weeks, the patient had complained of "gas pains" in the epigastrium coming on directly after meals and relieved by saleratus and pills given by his physician. There was no nausea, vomiting, diarrhea, constipation or specific intolerance to fatty or fried food. The patient had been working during these 2 weeks. On the morning of admission, he suddenly felt a general weakness and vomited about a pint of bright-red blood. He was immediately taken to the hospital, where he again brought up a glassful of bright-red blood.

The patient had had pneumonia 1 year previously and an acute upper respiratory infection 2 weeks previously that had since disappeared. He had had no dyspnea, orthopnea, cyanosis or edema. His mother had died of cancer. His wife and a son were well. A daughter died, at the age of 16, of heart trouble.

Physical examination showed a well-developed, fairly well-nourished man who was somewhat pale and extremely apprehensive. The temperature was 96°F., the pulse 78, and the respirations 20. The pupils were round, regular and equal, and reacted to light and accommodation. The scleras were clear. The conjunctivas were moderately pale. The mucous membrane of the mouth was of fair color. The tongue protruded in the midline, without deviation.

There was no stiffness or retraction of the neck. No lymph nodes were palpable. No dullness or rales were present in the lungs. The breath sounds were normal. The heart was not enlarged to percussion, and the sounds were of good quality. The rhythm was regular, with no murmurs. The blood pressure was 80/60. The abdomen showed no spasm or tenderness, and no masses were felt. The extremities were symmetrical, without ulcerations. The knee jerks were normal.

Examination of the blood showed a red-cell count of 2,600,000 with 54 per cent hemoglobin, and a white-cell count of 12,400 with 61 per cent neutrophilic leukocytes, 25 per cent lymphocytes, 4 per cent large mononuclears and 10 per cent polynuclear band cells. The red blood cells were hypochromic. The blood Kahn reaction was negative. The urine was normal.

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The diagnosis was bleeding peptic ulcer.

The patient's blood was typed. He was placed on a modified Meulengracht's[12] diet and aluminum hydroxide. He stopped vomiting, and his condition in the next several days improved rapidly. On the 7th hospital day, he developed auricular fibrillation, and dullness and diminished breath sounds in the left chest. A film taken with a portable x-ray apparatus revealed a massive collapse of the left lung. The right lung was clear. On the following day, shortly after midnight, the patient began to vomit huge amounts of bright-red blood, and died before any possible aid could be given.

Autopsy (8 hours post mortem). Gross examination showed blood in the mouth. In the descending portion

FIGURE 1. *Post-Mortem Specimen.*
The arrow marks the area of perforation of the aneurysm into the esophagus.

of the aortic arch was a saccular aneurysm, which extended from the 4th to the 9th thoracic vertebra and had eroded the left side of the 5th to 8th dorsal vertebral bodies. It was adherent to the lower portion of the esophagus at a point 5 to 7 cm. above the cardiac orifice of the stomach and also to the left main bronchus about 1 cm. below the bifurcation, and had extended through the walls of both these structures, forming oval protrusions into their lumens. The defect in the esophageal wall measured 3.5 by 2.3 cm., and the domelike elevation of the sac wall was gray except at its lower edge, where there was an opening from the sac into the esophagus, which was 5 or 6 mm. in diameter and admitted the tip of a forceps. A little blood was present in the esophagus. The extension of the aneurysm through the bronchial wall was similar but smaller.

The stomach was greatly distended by blood, much of which was clotted, and the rugae were flattened. The mucosa showed a mild congestion, but no ulcerations were seen in it or in the duodenum.

Microscopic study of the aorta and the wall of the aneurysm revealed many foci of new blood vessels and monocytes in the media and a variable widening of the adventitia in which an increase of fibrous tissue and an invasion of lymphocytes and plasma cells, especially around the blood vessels, were noted. Part of the mass lining the aneurysm consisted of acellular fibrin, frequently in laminated form, and part had a definite thrombus structure. Over the extensions into the bronchus and esophagus, the wall was very thin and consisted of only a few strands. The lung alveoli and the small bronchi contained no blood.

Anatomical diagnoses: syphilitic aortitis, with aneurysm of descending portion of arch, erosion of thoracic vertebras, erosions into the left bronchus and esophagus, and hemorrhage into the esophagus.

DISCUSSION

So-called "gas pain" in the abdomen experienced by the patient on a few occasions before admission to the hospital is suggestive of many conditions. It may be a sign of organic as well as functional disease. But when the patient also suffers an episode of severe hematemesis, grave organic disease is almost surely present. Bleeding peptic ulcer is the commonest cause of such symptoms. It is true that a number of other conditions may cause hematemesis — carcinoma or, more rarely, lymphoma or sarcoma of the stomach, aortic aneurysm, mesenteric venous thrombosis, carcinoma of the gall bladder eroding the duodenum, acute esophagitis, esophageal varices due to cirrhosis of the liver, abdominal trauma, thrombocytopenic purpura, carcinoma of the pancreas eroding the duodenum, acute gastritis and Banti's disease. However, such causes of hematemesis are comparatively rare.

Because the physical examination and laboratory findings were essentially negative, except for acute secondary anemia, in the case presented above, peptic ulcer was considered the most probable diagnosis. The patient responded very well to rest and diet, and his general condition was good. On the seventh day, he suddenly developed auricular fibrillation and atelectasis of the left lung. Because of these new findings, attention was focused on the chest, and for the first time there was some doubt. Was the bleeding from the upper gastrointestinal tract, lungs or great vessels? Unfortunately, the patient suddenly died the following day from the massive hematemesis, and there was no opportunity for further study.

SUMMARY AND CONCLUSIONS

A case of a syphilitic aneurysm of the descending portion of the arch of the aorta, with rupture into the esophagus, is reported.

Although aneurysm has long been recognized as a clinical entity, the silent forms are rather unusual and may present a difficult diagnostic problem, especially if one does not consider such a possibility. Surprise and humility of the attending physician can be prevented if each patient with a severe hematemesis of unknown origin is immediately fluoroscoped in a horizontal position; care should be taken to avoid all abdominal palpation. Thus, no possible harm can be done to the patient, and aneurysms of the aorta can be ruled out.

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MEDICAL PROGRESS

LICHEN PLANUS: DIAGNOSIS, ETIOLOGY AND TREATMENT

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ALTHOUGH lichen planus has for generations engaged the profoundest interest of physicians the world over, it remains a baffling problem and still constitutes a decided challenge to medical science. The disease is all the more absorbing in its interest because it represents such a clear-cut morphologic entity. It is an appealing and stimulating subject for investigation concerning both interpretation and treatment.

Of the neurogenic, toxic and microbic theories of causation, the first is probably the most popular among writers at present. However, very little convincing evidence regarding causation has accumulated, and no tangible clues to the specific etiology have been found. The treatment has not advanced beyond the empirical stage, and is still chiefly a matter of mercury, arsenic, bismuth and x-ray. However, the use of vitamin B complex, as recommended in a recent publication by Burgess,[1] may prove to be a worthy contribution. The results obtained by Burgess were so favorable as to suggest that his observations will lead to the discovery of some therapeutic method as effective as but less dangerous than some of the procedures now in use. Possibly, this improved form of treatment will be some fraction of the vitamin B complex.

The diagnosis of lichen planus is not difficult in most cases, and yet the behavior of the disease is so varied as to present occasional hazards. Certainly, it is not easy to learn to diagnose lichen planus by textbook descriptions or photographs any more than it is with many other skin diseases. Nevertheless, after one has studied with care a few typical cases, appropriate diagnostic acumen for this disease may be acquired.

The primary lesion of lichen planus is a shiny, flat-topped angular papule, which nearly always presents a violaceous tinge. This lesion is so characteristic that, if examined carefully, it is not likely to be confused with other primary lesions. The papules of lichen planus may develop in rows along lines of scratch. Favorite areas of distribution include the flexor surfaces of the wrists, the inner aspects of the legs and thighs, the penis and the mucous membranes of the mouth and lips. In contrast with the usual tendency toward angular morphology, lichen planus may take the form of ring-shaped lesions, which are encountered especially in the region of the wrists and genitalia. The mucous-membrane changes may be of the nature of silvery stippling, or a flat whitish lacelike network, or whitish patches and streaks. The mucous-membrane lesions may resemble leuko-

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plakia, with which lichen planus is often confused. In extent, the disease may vary from a few papules to involvement of practically the entire body surface. The expert must learn to recognize atrophic, hypertrophic, bullous and other forms of lichen planus. Pruritus may vary in degree from mild discomfort to intolerable agony, sometimes leading to suicide.

Lichen planus must be differentiated from other papulosquamous eruptions, such as psoriasis, parapsoriasis and secondary syphilis. No great difficulty should be encountered in distinguishing lichen planus from pityriasis rosea, tinea versicolor and seborrheic dermatitis. A typical case of lichen planus bears no real resemblance to psoriasis. On the other hand, it is worth remembering that many of the lesions in some of the borderline cases of lichen planus may be decidedly psoriasiform. Such lesions show layered, silvery scaling difficult to distinguish from psoriasis and a distinctive erythematous tint strongly suggestive of psoriasis. In psoriasis, the scales are usually thicker and more abundant, and bleeding points may be found. Psoriatic plaques are formed by peripheral extension of the initial papular lesions, whereas lichen planus patches are formed by coalescence of papules. In psoriasis, the elbows and knees seldom escape involvement; in lichen planus, the flexor surfaces of the wrists and the inner aspects of the legs and thighs are areas of predilection. Lichen planus is frequently seen on the mucous membranes of mouth and lips, whereas psoriasis is almost never found in these areas. Both conditions may involve the penis.

In patients who present a papulosquamous skin eruption resembling both psoriasis and lichen planus, the differentiation may remain quite difficult until the skin has been examined completely. Then it is usually possible to demonstrate diagnostic points that swing one's judgment decidedly one way or the other. The finding of characteristic lichen planus papules or typical mucous-membrane lesions quickly settles the matter.

It is advisable to perform blood Wassermann, Hinton and Kahn tests in every case of lichen planus, and only in this way can one be sure to avoid occasional errors in confusing this disease with papulosquamous secondary syphilis.

Lichen planus limited to the oral mucous membranes is not difficult to recognize because of its peculiar morphology, which consists in a whitish stippling, patches and streaks or a whitish lace-like network. Lichen planus of the oral mucous membranes is less likely to be confused with leukoplakia if there are lesions elsewhere in the skin.

Lichen planus may be limited in its distribution to the vulva and in this way becomes of considerable interest to surgeons, gynecologists and obstetricians, who must differentiate it from kraurosis vulvae and leukoplakia. Biopsy must be carried out at times, to aid in the differential diagnosis, particularly if there are no lesions elsewhere over the body to help in arriving at a decision. Because of this circumstance, the histologic picture of lichen planus is probably at times of even greater usefulness to gynecologists than it is to dermatologists.

Authorities agree that the papules of lichen planus possess a histologic structure that may be regarded as pathognomonic and permits accurate diagnosis from biopsy material. A characteristic feature is the limitation of the cellular infiltration to a regular band in the upper cutis.[2] The lower border of this lymphocytic infiltration is sharply outlined, and the upper border lies just subjacent to the basal-cell layer. Among other features of the histopathology, McCarthy[3] includes the following: well-defined, circumscribed (papular) lesions; hyperkeratosis, without parakeratosis; widening of the stratum granulosum; acanthosis, with formation of ill-developed rete cones; hazy outline between epithelium and corium; space formation at the cutaneous epithelial border; dilated blood vessels of the papillary and subpapillary layers; and occasional giant cells (Sabouraud).

Cases of both familial and conjugal lichen planus have been reported. It is of great interest to study these case histories, because of the light that they seem to cast on the etiology of this strange disease. The interpretation of lichen planus as a familial disease was apparently first suggested by Hallopeau and Leredde[4] in 1900, although several observers, including Lustgarten,[5] Brocq[6] and Ormerod,[7] had reported cases with a familial background.

Of approximately 60 cases of familial lichen planus reported in the literature, some of the most unusual have been summarized by Saffron[8] as follows: an acute attack that developed in a mother while she was treating a daughter who had the disease (Brocq[6]); hypertrophic lichen planus in three generations — mother, son and grandson (Joseph[9]); an acute form that developed in two sisters living apart, six weeks after the death of their father (Little[10]); the annular, atrophic variety in two sisters, with the lesions confined to the legs (Samuel[11]); acute lichen planus, in two brothers who were not living together, complicated by glandular and splenic enlargement (Bettmann[12]); typical lesions in a ten-month-old nursling whose mother had lichen planus (Wende[13]); lichen planus verrucosus in a father and son (Von Ken-

nel[14]); lichen planus in the father, son and daughter of a family of four (Veiel[15]); and three brothers who had lichen planus (Scheer[16]).

The occurrence of 4 cases of lichen planus in one family has been reported by Saffron,[8] who concludes that the familial occurrence is an argument for the neurogenic theory of the disease.

The family consisted of father, mother, four sons and a daughter. None of the children had lived with the parents or with one another for ten years. All members of the family were well educated, sensitive and of above average intelligence. The mother, at the age of fifty-two, developed acute lichen planus in 1921 at a time when, presumably, the children were living with the parents. Eleven years later, in 1932, one son, aged thirty-six, developed the disease. In 1936, fifteen years after the mother's first attack, another son, aged thirty-seven, developed lichen planus, and in this same year the mother had her second attack. In 1938, seventeen years after the mother's first attack, still another son, aged thirty-eight, had an acute widespread attack of lichen planus; in the following year, this son had a second attack. The mother's first attack lasted five months, and the second eight months. The first son's attack lasted three months, and the second son's six months. The third son's first attack lasted three months, and his second eight months. All attacks in this family were associated with severe pruritus except for the mild attack in the first son, who had only slight itching. The first son was a salesman, the second a dentist, and the third a factory manager.

Lichen planus of the oral cavity in twins has been reported recently by Epstein[17] as an occurrence previously unknown in the literature. Epstein interprets these cases as evidence favoring both the theory of familial predisposition and the theory of infection. However, his patients displayed indisputable nervous factors and, therefore, could be used in support of the neurogenic hypothesis. It is not illogical to presume that in some of the cases of lichen planus there may be an etiologic interplay of all three of these elements. Abstracts of the 2 cases are as follows:

A 25-year-old man was seen in January, 1939. He had noticed white lesions in his mouth for about 2 months. On examination, he presented numerous confluent reticular lesions covering nearly all the mucous membranes of the cheeks. The appearance was typical of lichen planus, and the additional finding of two annular lesions on the tongue helped to substantiate the diagnosis. In the course of 6 months, the patient consumed 60 cc. of potassium arsenite solution (*U. S. P.*), and the eruption cleared except for a few lesions. Treatment was then changed to vitamin B complex, and good condition was maintained so long as he continued vitamin B complex capsules. Each time he stopped taking these capsules, the disturbance showed a tendency to recur.

The identical twin of this patient was seen in February, 1941, 2 years following his brother. This patient began to notice lesions on the mucous membranes, the middle of the tongue and the lower lip about 8 months after his twin brother had begun to notice symptoms. The similarity of the eruption in the 2 cases was so striking that the patient himself made the correct diagnosis. Examination revealed a picture similar to that described in the case of his brother, with the added finding of a typical annular lesion on the lower lip. The patient treated himself by means of vitamin B complex in dosage up to 9 capsules a day. His condition improved greatly.

Interesting features of these 2 cases of lichen planus are the following: the patients were identical twins; no other members of the family were affected — neither their parents, with whom they lived, nor their brother, with whom they worked, had the condition; both patients were nervous and high strung and admitted hurrying and worrying all the time, and both observed that they were much better when not worried or excited; the clinical picture in the 2 cases was almost identical.

Similarity of symptoms in familial lichen planus has been reported too often to be considered a coincidence. Little's[18] observation of lesions confined to the mucous membranes of the mouth in mother and son corresponds to the cases reported by Epstein.

Spitzer,[19] in 1924, collected from the literature 46 cases of familial lichen planus, in 7 of which husband and wife were affected. Since 1924, 12 additional reports of familial lichen planus have appeared. Of these, only 3 have been conjugal in nature.[20–22]

Of the 10 known cases of conjugal lichen planus, the most recent was reported in 1937 by Goodman and Sulzberger,[20] who encountered this interesting example in their office practice.

The wife, aged 45, when first seen in August, 1935, gave a history of having had a pruritic skin disorder for 8 months. She stated that her husband had complained of itchy lesions on his legs for more than a year prior to the onset of her trouble. Examination disclosed a typical lichen planus consisting in widely distributed, purplish, flat-topped papules. Some of these had an annular grouping. The buccal mucosa showed a network of minute whitish lesions, and there were whitish patches involving the perianal and perivulvar areas. The findings were so typical of lichen planus as to leave no possible doubt regarding the diagnosis. The lichen planus cleared in large part in 3½ months following x-radiation, bismuth and mercury.

The patient's husband was a 53-year-old salesman who was seen, shortly after his wife's satisfactory recovery, in January, 1936. His lichen planus had been slowly extending during his wife's period of recovery under treatment. On examination, he showed typical hypertrophic lichen planus on the legs and ordinary lichen planus elsewhere. There were scattered angular flat-topped papules and occasional ringed forms. One large, annular lesion was present on the penis. Involvement of the lower lip

and buccal mucous membrane left no further doubt regarding diagnosis. This patient recovered satisfactorily following x-ray therapy and mercury. The hypertrophic patches were resistant at the start, but later they were reduced considerably by applications of trichloracetic acid.

The occurrence of conjugal lichen planus seems to be an argument for the theory of infection.

No discussion of the etiology of lichen planus could be considered adequate without further mention of the theory of infection and the evidence for it. In 25 out of 28 typical cases, Jacob and Helmbold[23] isolated a gram-negative anaerobic bacillus, morphologically resembling those of the colon-typhoid group, which grew freely on a semisolid glucose human-serum medium containing pieces of human tissue. In sections of the lesions, they were able to demonstrate the organism but could not do so in normal skin or in other types of papular skin disorders. By inoculation of the skin of normal persons with this organism, they occasionally succeeded in producing lesions that resembled lichen planus both clinically and histologically.

According to Guttmann,[24] modern work has failed to show any connection between the nervous system and lichen planus. Other observers, however, including Ormsby,[25] Pusey,[26] MacLeod[27] and O'Donovan,[28] believe that the disease can be precipitated by mental disturbance. Ramel[29] has reported the case of a woman who developed lichen planus a few days after severe mental trauma; she resisted the usual forms of treatment but recovered following psychotherapy, which brought to consciousness a typical Freudian complex. Eller[30] observed that, during a serious fall in the stock market, acute lichen planus developed among a number of business men who had suffered financial losses. The wife of one of these was affected similarly.

To determine whether certain dermatoses occurred more frequently among nervous patients than among the general population, Goldsmith[31] compared their relative incidence in proportion to the total number of new patients attending the West End Nerve Hospital with their relative incidence in proportion to the total number of new patients attending a general hospital, the University College Hospital. Lichen planus was found by Goldsmith to be only half as common at the nerve hospital.

Culver[32] believes that there is no nervous element in the etiology of lichen planus and quotes in support of this the lack of cases in California during World War I. Since the disease is not reportable, no figures are available to prove whether war actually increases, decreases or fails to affect the incidence. Usually, it is assumed that there has been no increase in lichen planus during wartime and that this constitutes an argument against the theory of nervous origin. On the other hand, it might be reasoned that war creates a type of stress and strain that crowds out minor financial or family worries and perhaps paves the way for a type of adjustment that does not tend to precipitate attacks of lichen planus.

The generally recognized modern therapeutic approach to lichen planus consists essentially in mercury, arsenic, bismuth and x-ray therapy, as previously mentioned. Other methods of treatment include psychotherapy, injections of extracts of the papules, foreign-protein therapy with colon typhoid vaccines or milk, and lumbar puncture. Local applications are useful only as antipruritic agents. Stelwagon[33] has advised that "the patient is to have the benefit of good, plain food, hygienic living, and, when possible, outdoor life and freedom from mental worry or care."

When the disease is extensive, all forms of injection therapy have an advantage over x-radiation. The bismuth therapy of lichen planus has the advantage that it is less apt to be complicated by untoward reactions than arsenic and mercury are. Bismuth may be administered as the salicylate in oil by intramuscular injection in doses of 1 or 2 cc. at weekly intervals, as in the treatment of syphilis. Bismuth is less effective than arsenic but is advisable for use at the start of treatment because its complications are fewer. Arsenic may be substituted if the therapeutic response to bismuth is inadequate.

The French preparation, Énésol, has been very popular in this country for the treatment of lichen planus and has been considered one of the most reliable forms of therapy. This drug, which is a combination of mercury and arsenic, is administered intramuscularly in doses of 1 or 2 cc. once or twice weekly. Eight to sixteen or more injections may be necessary. The usual reactions to mercury and arsenic must be looked for.

Conrad et al.[34] have reported 25 cases treated by the intramuscular injection of bismuth arsphenamine sulfonate (Bismarsen). They employed twelve to twenty-five injections of 0.1 to 0.13 gm. each. Patients showing signs of a mild toxic reaction to Bismarsen — skin changes, drowsiness, stomatitis and puffiness of the legs — were given large dosage of vitamin C while the drug was continued, and all these reactions disappeared. In some cases, the drug had to be stopped owing to severer reactions. Conrad and his associates treated hypertrophic, localized and generalized types of lichen planus and concluded conservatively that Bismarsen seems to be of value.

Sulzberger[35] recommends that arsenic be administered in the form of a 2 per cent sodium ar-

senate solution containing 1 or 2 per cent phenol. This solution, prepared with sterile water and placed in a sterile container, should be kept in the refrigerator. Injections are given daily by the subcutaneous route. The initial dose is 2 minims, which is increased by 1 minim daily until a dose of 20 to 30 minims (1.5 to 2.0 cc.) is reached. If the eruption disappears or if signs of intolerance develop, the arsenic should be stopped. If necessary, the dosage may be increased to 60 minims (4.0 cc.) daily, and then given in decreasing daily doses until improvement takes place.

Fowler's solution may be given by mouth if the daily subcutaneous injection is not expedient. Sulzberger[35] recommends a mixture containing 1 part of Fowler's solution and 2 parts of compound elixir of pepsin (*N. F.*) or menthol water. Administered in this form, Fowler's solution is more palatable and less irritating to the gastric mucosa. The dose is 10 to 30 drops (0.6 to 2.0 cc.) of the diluted solution three times daily, following each meal.

Arsenic may be administered by mouth in the form of the Asiatic pill (arsenic trioxide). This pill is available in three strengths: 0.002 gm., 0.004 gm. or 0.007 gm. arsenic. At the start, the weakest pill is given three times daily. Later, the dose is increased to the limits of tolerance.

Treatment for four to six weeks is usually necessary before the effects of arsenicals are visible. The treatment, if successful, will bring about relief of itching and disappearance of the lesions, leaving only residual pigmentation and occasionally depigmentation.

In a series of cases of lichen planus some of which had defied the usual treatment for years, Biberstein[36] succeeded in curing 70 per cent by injecting extracts of the papules.

Recent studies by Burgess[1] on the use of vitamin B complex in the treatment of lichen planus are of great interest from the standpoint of both etiology and therapy. His encouraging results are also well worthy of attention because they came about as a consequence of the use of a type of treatment bearing no serious hazards. Burgess points out that the majority of dermatologists have resorted to the theory of neurogenous causation in preference to the parasitic, microbic and toxic. Both he and Goldsmith[31] cite Ramel,[29] who found that violent emotional shock produced the eruption in some cases and that cure resulted from psychoanalytic treatment. Burgess believes that the good results obtained from lumbar puncture and irradiation of certain segments of the spinal column may be ascribed to the psychic value of these procedures. In keeping with these lines of reasoning, Burgess then set out to study the therapeutic effects of vitamin B complex on the theory of its possible action in relieving the nervous tension of these patients.

Some of the cases studied by Burgess may be briefly abstracted as follows:

Case 1. Treatment was carried out by means of thiamine chloride given by mouth in daily dosage of 3000 international units and liver extract administered intramuscularly twice weekly in doses of 2 cc. There was lessened irritability in a few days and beginning involution of the lesions in 3 weeks.

Case 2. Liver extract, given by intramuscular injection in doses of 2 cc. every 2 days, brought about marked regression of lesions in 1 week.

Case 3. Beminal was given by mouth in daily dosage of 120 cc. and nicotinic acid in daily dosage of 150 mg. Striking regression took place in 4 days.

Case 4. Marked involution took place in 3 weeks following the use of Beminal by mouth and liver extract by injection.

Case 6. The response was precisely the same as that in Case 4.

Case 7. After 6 years' duration, recovery took place in 6 weeks following the use of Beminal and liver extract by mouth.

Case 8. After 3 years' involvement of the legs and mucous membranes, marked improvement took place in 1 month following the use of Beminal and liver extract by mouth. Complete recovery took place in 2 months.

Case 9. After 2 years' involvement of the mucous membranes only, complete disappearance of lesions took place in 1 month following the use of brewers' yeast and nicotinic acid by mouth.

Case 10. A case of 4 months' duration, with involvement of the buccal mucous membrane and tongue, did *not* respond to yeast, Beminal, riboflavin, nicotinic acid, thiamine chloride, x-radiation, arsenic and mercury.

Case 12. Lichen planus developed in the course of antisyphilitic therapy. Brewers' yeast was given by mouth and thiamine chloride by injection. In 1 month, all lichen planus papules were gone.

Case 14. A case of lichen planus of the dorsal surfaces of the hands of 2 months' duration, with onset following a heart attack, did not respond to vitamin B complex and x-radiation.

Case 15. Hypertrophic lichen planus located on the inner aspect of the right knee showed improvement with x-radiation but, after 2 months of therapy by means of Beminal by mouth and liver extract by injection, the thickening disappeared completely and was replaced by an area of atrophy.

Burgess states that, in his series of 25 cases, vitamin B complex as a whole, rather than any special constituent, seemed to be effective. He found that such therapy was much less successful if mental unrest continued, and was more effective if nervous stress and strain could be relieved by rest and reassurance.

The itching of lichen planus may constitute a serious symptom; however, it may be entirely absent. The control of pruritus is in some cases beyond the powers of medical science. All forms of therapy known to be of value in bringing about

involution of the lesions are also of assistance in combating the subjective symptoms of this disease. Chloral hydrate is the safest soporific to employ. Sedatives such as phenobarbital may be used, but morphine should be avoided if possible, because of the tendency toward habit formation and its powers of aggravating the itching.

Topical applications in lichen planus are of no value except to help control discomfort. Antipruritic remedies may be made up in aqueous or alcoholic solution or in a vanishing-cream base, and they may contain 0.25 to 1 per cent menthol, 0.25 to 2 per cent phenol, 0.25 to 5 per cent camphor, 0.25 to 1 per cent thymol and 1 to 5 per cent chloral hydrate.

* * *

By full use of all that is known concerning the therapy of lichen planus, gratifying results may be expected in many cases. Some of the patients, however, resist all the usual procedures and suffer from the disease in unabated form for years. There is great need for a more detailed and critical study of these cases from the standpoint of psychiatry, clinical pathology and general medical investigation. There has been scant effort to refute or substantiate the work concerning microbic origin. If the cases were more carefully studied from the historical viewpoint, it is likely that more reports would come forth regarding familial and conjugal incidence, and the interpretation of such cases might become more clear-cut.

Lichen planus has many ramifications through the general field of medicine and, as mentioned above, may be limited in its distribution to the mucous membranes of the vulva; it thus becomes of special interest to surgeons, gynecologists and obstetricians. It may appear as a reaction to drugs such as arsenic and the heavy metals, the same chemicals so popular in its therapy. Obviously, it is not purely an external skin disturbance, but a constitutional illness of extraordinary character. Perhaps, the pathologic physiology of lichen planus will not be understood until more is known about what the activities of the sympathetic and parasympathetic nervous systems can do to tissues.

Burgess's[1] work on vitamin B therapy, which gives an entirely new angle to this many-sided subject, may provide a more satisfactory form of treatment, for which there has been a great need as voiced by many dermatologists at the recent meeting of the Academy of Dermatology and Syphilology in New York City. The work of Burgess should be repeated carefully because it really seems "too good to be true." If it is true, one must find out why vitamin B complex is effective, and it might be of value to investigate whether there is some vitamin B complex fraction of particular value.

A fuller understanding of lichen planus from the neurogenous angle might add a chapter to the rapidly increasing understanding of what the stresses and strains of life can do to the human body, and how these stresses and strains can be offset by vitamins, rest, psychotherapy and other measures.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

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CASE 28251

PRESENTATION OF CASE

First admission. A fifty-five-year-old Italian barber entered the hospital for study.

Three years before entry, he had an attack of "rheumatism," which soon disappeared. One year before admission, he noticed that dribbling occurred after urination and that his urine, which had not changed in appearance, caused itching of the skin of his legs and penis. This was accompanied by nocturia (once a night) but no other symptoms. Rheumatism recurred nine months before admission, and was characterized by intermittent pain and stiffness of the hands, shoulders, knees and left ankle. The symptoms, which were worse in the morning, were occasionally accompanied by swelling of the joints, with questionable reddening, but no incapacitation; they disappeared in six months. Meanwhile, the pruritus increased, was worse at night, and involved the penis, legs, abdomen and arms, but not the hands. No treatment was given, and relief was spontaneous one month before admission. The patient stated that his wife had noticed some pruritus three months before. Six months before entry, the patient lost his initiative and began to tire easily, and three months later noticed that he felt dizzy if he remained on his feet too long. This symptom finally forced him to stop work. One month before admission, he consulted his physician, who found a positive blood Hinton reaction and started treatment two weeks later. During this time, the patient suffered from an increasing thirst, a nocturia of two or three times a night and a steadily increasing sense of fatigue. Two days before admission, he noticed that his stools had become light brown, and a generalized pruritus developed; there was no jaundice. In addition, there were anorexia and postprandial distention. The patient had lost 8 pounds in an unstated period, and for the previous three months had had slight epistaxes after blowing his nose.

Thirty-seven years before admission, he had a penile chancre, which was treated locally. The family history was irrelevant.

On examination, the patient was well developed and somewhat undernourished, and the skin was believed to be more deeply pigmented than is usual for his race. The buccal mucous membrane was pale, and on the right there was a pigmented gray-black area. A generalized lymphadenopathy was present, with small, shotty epitrochlear, cervical, axillary and inguinal lymph nodes. The heart was not enlarged, and there was a soft blowing murmur at the apex. Examination of the lungs was negative. The liver was just palpable, and a firm, nontender spleen was felt four finger-breadths below the costal margin. There was a suggestion of atrophy of the hands, and the left hand, right foot and right knee were tender on motion and pressure.

The temperature, pulse and respirations were normal. The blood pressure was 150 systolic, 70 diastolic, sitting, and 130 systolic, 70 diastolic, standing.

Examination of the urine was negative. The blood showed a red-cell count of 5,530,000 with a hemoglobin of 65 per cent, and a white-cell count of 2800 with 33 per cent polymorphonuclears, 57 per cent lymphocytes, 2 per cent eosinophils and 8 per cent monocytes; the platelets appeared to be normal. The sedimentation rate was 0.48 mm., the serum van den Bergh was normal, and the blood Hinton and Wassermann reactions were positive. A lumbar puncture revealed nothing abnormal, and the spinal-fluid Wassermann reaction was negative. X-ray films of the chest were negative except for an area of calcification in the right infraclavicular region and questionable areas of calcification at the right hilum.

The patient was placed on an arthritic regime and given 5 cc. of Pentnucleotide. During hospitalization, the white-cell count did not rise above 3900, and for this reason it was deemed inadvisable to institute arsenical antisyphilitic treatment. He was discharged three weeks after admission under the care of his physician.

Second admission (four years later). A year after discharge, the patient was seen in the Out Patient Department complaining of generalized weakness, fatigue, joint stiffness and "gas on the stomach." Physical examination was negative except for a coarse tremor of the hands and hyperactive knee jerks. A month later, he returned complaining of swelling, tenderness and pain on motion in the hands and ankles, with pain in the hips and back after standing for a long time. Nocturia had increased to four times a night. The joints of the hands, knees and ankles were thickened and stiff, with tenderness on lateral pressure. Tenderness was also present over the sacroiliac joints and lower spine. A search for foci of infection revealed clear sinuses and chronic tonsillitis; the prostate was not enlarged, but moderately tender, and a smear contained many white blood cells. The patient was given a course of prostatic massage, and in a year the urine cleared and the

frequency subsided. Up to this point, he had had only one course of bismuth; two years before admission, the blood Hinton and Wassermann reactions were still positive. A course of neoarsphenamine was started by his physician, but after three treatments the patient complained of upper abdominal distress, gaseous eructations, fatigue, constipation and clay-colored stools; he developed jaundice and a palpable liver, with tenderness in the right hypochondriac region. The red-cell count was 6,430,000, and the white-cell count 6100 with 47 per cent polymorphonuclears, 43 per cent lymphocytes, 6 per cent eosinophils, 3 per cent monocytes and 1 per cent basophils. The patient was seen in the Out Patient Department two weeks later, when jaundice was still present and the liver was just palpable and slightly tender.

Six weeks before admission, the patient contracted a cold followed by a discharging right ear. He was admitted to the Eye and Ear Infirmary, where examination revealed a right otitis media and mastoiditis; culture of the aural discharge yielded hemolytic streptococci. There was a fine nystagmus to the right. An ulceration was present in the left hard palate, with tender, enlarged cervical lymph nodes. The spleen was enlarged and firm and extended almost to the umbilicus, and there was tenderness in the right upper quadrant. The red-cell count was 3,820,000 with a hemoglobin of 61 per cent, and the white-cell count was 3800 with 58 per cent polymorphonuclears and 42 per cent lymphocytes. Repeated lumbar punctures, including spinal-fluid Wassermann reactions, were negative, but a blood Hinton reaction was positive. On the ninth hospital day, a right simple mastoidectomy was performed, and one week later, the patient was transferred to the general hospital. It appeared that six weeks before admission, after five bismuth injections administered by his physician, a few red nonpruritic, slightly raised papules appeared on the flexor surfaces of both arms and spread to involve the legs in the course of the next week. The bismuth was discontinued, but the papules increased in number and appeared on the face. In the preceding few days, the lesions had faded somewhat.

On examination, the patient was wasted and sick. A diffuse, brownish-red, papular eruption, each papule measuring approximately 0.5-cm., was present on the skin of the forearms, palms, buttocks, knees and calves, but no lesions were seen on the mucous membranes. There was a generalized lymphadenopathy. Examination of the heart and lungs was negative. The spleen was palpable 3 cm. below the level of the umbilicus, and the liver one fingerbreadth below the costal margin. There was some stiffness and limitation of movement in the fingers of the right hand. Examination of the nervous system was negative.

The temperature occasionally rose above normal, the pulse averaged 95, and the respirations were normal. The blood pressure was 120 systolic, 80 diastolic.

Examination of the urine was negative. Examination of the blood showed a red-cell count of 3,800,000 with a hemoglobin of 65 per cent, and a white-cell count of 4200 with 10 per cent polymorphonuclears, 46 per cent lymphocytes, 40 per cent monocytes and 4 per cent eosinophils; the platelets were normal in number. The red cells were microcytic and hypochromic, with much variation in size and shape; no immature cells were found. The sedimentation rate was 0.92 mm., and a hematocrit reading 31.5; the bleeding and clotting times were normal, and an erythrocyte fragility test was normal. The nonprotein nitrogen of the blood serum was 20 mg. and the fasting blood sugar 90 mg. per 100 cc. The serum van den Bergh was normal, a bromsulfalein test negative, and a formol-gel test positive; the blood vitamin C level was 0.34 mg. per 100 cc., the plasma prothrombin 95.2 per cent, and the antithrombin 117 per cent. Blood Hinton and Wassermann reactions were positive. A tuberculin test was negative with a dilution of 1:10,000; a Congo-red test showed 63 per cent retention in the serum in one hour. Examinations of the stools were negative.

An x-ray film of the chest was negative. The knees were normal; the hands showed tufting of the terminal phalanges and arthritic changes about the joints consistent with rheumatoid arthritis. There was no periostitis to suggest pulmonary osteoarthropathy. A gastrointestinal series showed slight irregularity in the lower end of the esophagus, but varices could not be demonstrated. The stomach was displaced to the right by a lobulated mass, said to be a markedly enlarged spleen. The liver shadow appeared smaller than usual. X-ray study of the pelvis showed evidence of osteoporosis of the visible bones, but no other evidence of disease. There was a slightly fusiform area of calcification projecting into the true pelvis on the left side. Films of the femurs showed a very small area of calcification in the left popliteal space. There was no evidence of Gaucher's disease.

A biopsy of a lymph node showed chronic inflammation and pigmentation. A skin biopsy showed a rather marked perivascular infiltration containing many plasma cells, a picture consistent with parapsoriasis lichenoides.

During hospitalization, the rash gradually faded, and the mastoid incision healed satisfactorily. Frequent slight epistaxes were noticed. The white-cell count varied between 4200 and 1550. One blood film showed 22 per cent polymorphonuclears, 56 per cent lymphocytes, 4 per cent monocytes, 8 per cent eosinophils, 2 per cent basophils and 8 per cent myelocytes; another film showed 4 per cent myelocytes. The patient was discharged one month after admission to the Out Patient Department.

Final admission (five months later). The patient returned to the Out Patient Department three months before admission. His strength had returned to a certain degree, but he complained of finger stiffness that was worse in the morning and of inability to flex the fourth and fifth left fingers. Splenomegaly, hepatomegaly and generalized lymphadenopathy were again noted, and there was slight clubbing of the fingers and edema of the legs. The red-cell count was 4,740,000 with a hemoglobin of 72 per cent, and the white-cell count 2600 with 12 per cent polymorphonuclears, 60 per cent lymphocytes, 16 per cent monocytes, 4 per cent eosinophils and 8 per cent basophils; the sedimentation rate was 1.04 mm. The patient was examined again seven weeks later and felt stronger, but showed the same physical findings except for the disappearance of peripheral edema. One month before entry, he was admitted to the Eye and Ear Infirmary complaining of general malaise, anorexia, fever, loss of weight and a mass on the right side of the jaw that had made its appearance five weeks previously. This mass was an abscess and was incised and drained, yielding purulent material that contained hemolytic streptococci and *Staphylococcus aureus*. Another area of redness and swelling that developed below the major abscess was drained, and a biopsy of the surrounding tissue showed chronic inflammation consistent with gumma. The patient was transferred to the general hospital. It appeared that he had had fairly frequent small epistaxes for the previous two years. In the four years of observation, syphilitic treatment had consisted of twenty-two injections of bismuth and nine injections of arsenicals, with mercury and potassium iodide from time to time. Each attempt was hindered by unfavorable systemic reactions, which included jaundice, pruritus, diarrhea and skin reactions. The patient had lost 34 pounds in an unstated period.

On examination, the patient was emaciated but in no discomfort, and the skin showed a more than racial pigmentation. Over the arms and forelegs were several depigmented scars about 1.5 cm. in diameter, with areas of hyperpigmentation over the legs. The right buccal mucous membrane showed an area of deep-brown pigmentation. A draining sinus with surrounding induration was present in the right neck below the mandible. The scleras appeared slightly icteric. There was a generalized, shotty, painless lymphadenopathy. Examination of the heart and lungs was negative, except for a soft blowing apical systolic murmur. Examination of the abdomen revealed a smooth, firm spleen, which was not tender and reached to one fingerbreadth below the umbilical level. The liver could be palpated one fingerbreadth below the costal margin in the midclavicular line and three fingerbreadths below the xiphoid; it was not tender, and the edge was smooth and sharp. Ascites could not be demonstrated. There was clubbing of the fingers and toes, and the patient was unable to flex the right second, third and fourth fingers completely.

The temperature spiked to 101°F., the pulse was 90, and the respirations were normal. The blood pressure was 108 systolic, 70 diastolic.

Examination of the urine showed a ++ test for albumin. Examination of the blood showed a red-cell count of 4,750,000 with a hemoglobin of 65 per cent, and a white-cell count of 1800 with 1 per cent myeloblasts, 0.5 per cent premyelocytes, 2 per cent myelocytes, 7.5 per cent juveniles, 5 per cent band cells, 4.5 per cent segmented polymorphonuclears, 0.5 per cent large lymphocytes, 58 per cent small lymphocytes, 16.5 per cent monocytes, 3 per cent eosinophils, 1.5 per cent basophils, 1 per cent microblasts and 1.5 per cent Turk cells; the platelets appeared normal. A glucose-tolerance test gave a fasting level of 131 mg., in half an hour 188 mg., in one hour 228 mg., and in two hours 123 mg. per 100 cc. A blood Hinton reaction was positive, and a blood culture negative.

The patient continued to run a fever up to 101.6°F., and an area of fluctuant redness developed beneath the large abscess; within two days, the temperature was 104°F., but the white-cell count remained around 2000 despite the administration of Pentnucleotide. On the eleventh hospital day, the abscess was drained, and sternal-marrow and skin biopsies taken. At the end of this procedure, the blood pressure was 80 systolic, 69 diastolic, the pulse 200, and the respirations 40. That night, the patient had a chill, and the next day failed rapidly, developing coarse bubbling rales and wheezes throughout both lungs, with signs of consolidation at the right base, marked dyspnea and cyanosis, and shifting dullness in the abdomen. He died that day, the twelfth after admission, despite supportive measures and two 500-cc. blood transfusions.

A biopsy of the sternal marrow showed a pronounced increase in cellularity, with a preponderance of erythrogenic forms, among which normoblasts predominated, although there were signifi-

cant numbers of erythroblasts. There were increased numbers of eosinophils and no evidence of obstruction of granulocytic maturation. Megakaryocytes were normal in number. The skin biopsy showed chronic inflammation, and although the appearance was not that of gumma, syphilis could not be absolutely ruled out.

Differential Diagnosis

Dr. John H. Talbott: There are several diagnoses to be considered in this case. From the duration of symptoms, I think we are justified in assuming that the patient had a serious and chronic malady. The initial complaints of itching, frequency and dribbling suggest the possibility of diabetes mellitus. This supposition is strengthened by the findings of pigmentation, hepatomegaly and splenomegaly, and one might consider hemochromatosis. The glucose-tolerance curve showed a delayed response, not unlike that seen in many types of chronic disease. The urine was negative for sugar at all times, however. I do not believe the patient had either diabetes mellitus or hemochromatosis, and I mention them only to discard them. We are cautioned, on the other hand, that liver disease was present. The fact that an arsenical reaction followed the administration of arsenic, even in the absence of jaundice, points toward a hepatitis. Addison's disease and Gaucher's disease should be mentioned in passing as improbable causes of the pigmentation. The data regarding the hepatomegaly and the splenomegaly are impressive. The lower edge of the spleen was felt four fingerbreadths below the costal margin and terminally extended below the umbilicus. This was a large spleen such as may be associated with myelogenous leukemia or Banti's disease.

The leukopenia further points to a blood dyscrasia. The white-cell count is known to have risen above 5000 on only one occasion during the four-year period, following an acute infection. At times, it was as low as 1500, with a marked diminution in polymorphonuclears, an increase in monocytes and, terminally, an increase in myelocytes. The leukopenia did not respond to Pentnucleotide administration. Several causes are suggested for the leukopenia. The ulcer in the mouth, following an otitis media and mastoiditis, might make one think of agranulocytic angina. This was seen rather often a decade ago, and in many cases was attributed to the ingestion of amidopyrine. Amidopyrine poisoning must be discarded because we have no evidence that the patient ingested any of the material. The sulfonamides may produce a leukopenia, but again there is no history of ingestion. Finally, cirrhosis of the liver is associated with a persistent leukopenia.

The diagnosis of syphilis, I am sure, we must accept. A penile chancre was treated only locally, and persistently positive blood Hinton and Wassermann reactions were reported. The biopsy was consistent with gumma. The postarsenical hepatitis, which was observed on the second admission, four years after the first symptom, might be explained by one of several mechanisms. An arsenical hepatitis may develop in an essentially normal liver as an idiosyncrasy. Secondly, a hepatitis may be superimposed upon a Laennec's cirrhosis of the liver. Finally, a hepatitis may follow the use of arsenicals in a patient with a syphilitic cirrhosis.

The positive formol-gel test, an index of increased globulin content of the serum, confirms the impression that some liver disease was present, although the test may be positive in chronic infections, rheumatoid arthritis and syphilis. The Congo-red test was not sufficiently positive to justify a supposition of generalized amyloidosis with liver involvement. A normal person should retain at least 80 per cent of the dye in the serum; a typical case of amyloid disease retains less than 20 per cent. The clubbing of the fingers is interesting. There was no x-ray evidence of periostitis to suggest pulmonary osteoarthropathy. Clubbing is present in 10 or 15 per cent of patients with cirrhosis of the liver. I believe in this case we can attribute the clubbing to hepatic cirrhosis. Diffuse pigmentation may also be seen in cirrhosis. The pigmentation may be so extensive as to make the diagnosis of hemochromatosis an academic point.

The increase in size of the left lobe of the liver intrigues me. McCrae and Craven[1] reported several cases of syphilitic hepatitis with predominant regeneration in the left lobe. The statement in the abstract that the right lobe of the liver was below the costal margin and the left lobe was three fingerbreadths below certainly is suggestive of a syphilitic liver.

Disseminated lupus is a possible diagnosis in this case. The skin rash, a frequent finding in lupus, was probably due to bismuth in this patient. The arthritis and perivascular infiltration are seen in lupus. Leukopenia is also seen, but no mention is made of endocarditis, polyserositis, a persistent anemia or urinary abnormalities. Before seriously considering a diagnosis of disseminated lupus, I think we should have more leading data than we have.

What diagnoses are we willing to make? We have considered and discarded diabetes mellitus, hemochromatosis, disseminated lupus and Addison's disease. I believe that the patient had pneumonia, terminally. I believe that he had syphilis.

The blood serologic findings were positive, and a biopsy showed changes characteristic of a gumma. There was probably an arsenical hepatitis superimposed on a diseased liver. I cannot make a diagnosis of syphilitic hepatitis or cirrhosis. The clinical findings are consistent with rheumatoid arthritis. The intriguing points concern the liver and spleen. At least, they were the points that gave me the most concern. A syndrome is described in the literature that is presumably an atypical form of rheumatoid arthritis, or adult Still's disease, that is known as "Felty's[2] syndrome." This syndrome is associated with arthritis, even ankylosis of joints, leukopenia, cirrhosis of the liver and splenomegaly. It seems to fit the evidence best, and I shall rest my argument on this diagnosis.

Dr. Francis R. Dieuaide: One point of considerable interest, for which I cannot offer a good explanation, is the marked and fairly generalized lymph-node enlargement. So far as syphilis of the liver is concerned, I am unwilling to make such a diagnosis. One can rarely make that diagnosis with confidence when distortion and enlargement of the liver cannot be felt clinically. The cases that McCrae and Craven described were all of that nature, and these are the cases in which therapy seems to be effective. Some cases of syphilis of the liver do not show such distortion. They are uncommon, and the general appearance is almost exactly that of alcoholic cirrhosis of the liver both clinically and in the gross, post mortem. The marked enlargement of the spleen does not fit well with syphilis of the liver, unless one supposes that there was also syphilis of the spleen. The splenomegaly was probably due to venous obstruction. I believe that this patient had latent syphilis, and that syphilis played no important part in the illness.

Dr. Austin W. Cheever: I agree with you. I do not see anything pointing definitely to active syphilis. I am a little loath to accept the idea of bismuth dermatitis. Of course, it does occur. We see it very rarely.

Dr. John W. Zeller: This patient was seen in the arthritic clinic several times, and during the first admission Dr. Walter Bauer and Dr. A. O. Ludwig saw him and thought the symptoms could be explained on the basis of rheumatoid arthritis, a Felty's syndrome, although joint involvement is usually much more marked in such cases. At the next admission, there had been little progression in the joint involvement; the spleen was then so large and the leukopenia so great that they were inclined to believe the fundamental diagnosis must be something else.

Dr. Tracy B. Mallory: They did not commit themselves on that point?

Dr. Zeller: No; they wondered about cirrhosis of the liver but thought the evidence was not sufficient.

Dr. Dieuaide: The leukopenia should be emphasized; the differential count is of a great deal of interest and may well contain a clue that we do not get otherwise.

Clinical Diagnoses

Felty's syndrome.
Latent syphilis.
Gumma of right neck?
Abscess of right neck?
Septicemia?
Pneumonia or atelectasis of right lower lobe?

Dr. Talbott's Diagnoses

Felty's syndrome (atypical rheumatoid arthritis, cirrhosis of liver, splenomegaly).
Terminal pneumonia.

Anatomical Diagnoses

Cirrhosis of liver, portal, type undetermined.
Splenomegaly.
Bronchopneumonia, bilateral.
Lung abscess, multiple.
Pulmonary edema.
Arteriosclerosis, coronary and aortic, moderate.
Syphilis, serologic.
Ulceration of skin, right neck.
Aberrant renal artery, right.
Operative wound: sternal biopsy.
Degenerative arthritis.

Pathological Discussion

Dr. Mallory: The autopsy findings can be fairly briefly disposed of, and we can still continue to argue. We found a severe degree of cirrhosis of the liver. The liver was somewhat enlarged—weighing 2100 gm.—and was rather grossly nodular. There were no deeply pitted scars,—nothing that would permit a gross diagnosis of syphilitic hepatitis, and we did not consider that probable. Microscopic examination added very little. There was no hyaline degeneration of the alcoholic type, nothing to suggest gummas, and I must call it an idiopathic portal cirrhosis—I believe a very chronic one. I doubt if it resulted from the arsenic therapy, and I imagine that the patient had cirrhosis at the time he was first given arsphenamine. That, however, is open to question, and is no more than a guess. The spleen was large—weighing over 1500 gm.—and was rather tough and fibrous. On microscopic examination, it showed all the characteristic appearances of the spleen of severe portal congestion or Banti's disease. There were dilatation of the sinusoids, disappearance of cellular elements from the pulp and

replacement of the cords by fibrous tissue. There was also progressive fibrosis of the malpighian corpuscles and hemorrhage about them. All the changes found in the spleen are characteristic of those found in cases of severe portal obstruction. There was generalized lymph-node enlargement, and on microscopic examination those lymph nodes showed markedly dilated sinuses, increased vascularity, but comparatively little actual lymphoid hyperplasia. The size of the spleen and the degree and the persistency of leukopenia in this patient are perfectly consistent with cirrhosis of the liver. On the other hand, I do not believe there is any way on anatomic grounds that I can rule out Felty's syndrome. The patient may have had that as well, but the actual anatomic findings certainly suggest that the primary and important cause of death was cirrhosis of the liver.

DR. ZELLER: Dr. Granville A. Bennett received one finger for study, and the report we received from him on the histologic studies of the joints was that there was nothing in the joint to indicate a diagnosis of rheumatoid arthritis. He made a diagnosis of degenerative arthritis, but regretted that he had not received other joints for study.

DR. TALBOTT: Of course, one finger does not exclude rheumatoid arthritis. To exclude an arthritis, one would have examined many joints, and this was not done.

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CASE 28252

PRESENTATION OF CASE*

A four-week-old-female infant was admitted to the hospital because of weight loss and diarrhea of ten days' duration.

The patient was born by breech delivery at full term after an uneventful gestation period. She was in good condition at birth and suffered no complications during the neonatal period. She was formula fed from the outset, but had regained her birth weight—5 pounds, 3 ounces—at the time of discharge from the hospital when twelve days of age. She received an adequate intake of essential supplementary dietary factors. On arrival at home, she began to have an almost constant discharge of feces. The stools were not watery, but consisted of thin, ribbonlike, well-formed, uniformly consistent, greenish or yellowish material, which, on occasion, was blood streaked, especially during the week prior to entry. No gross blood, pus or mucus was noted. Accompanying the frequent bowel movements were episodes of "spasm," consisting of straining movements with flexed thigh, a shrill cry and congested, cyanotic facies. These spasms tended to occur only at night. The patient did not seem unduly irritable, slept well, and took feedings well. However, in spite of this and several changes of formula, the addition of "loose" and the reduction of fat content of the diet, the diarrhea persisted, and the infant lost 1 pound in weight, from 6 to 5 pounds, during the week before entry. She vomited only twice during her illness.

A sister and a maternal aunt of the patient had "allergic disorders."

On admission, physical examination was essentially negative except for slight pharyngitis, a mild rectal prolapse with slight bleeding, and the finding of marked vasomotor instability, with rapidly alternating pallor and flushing of the skin, and mottling and cyanosis during crying spells, variations in heart rate, with a predominating tachycardia of 150 to 200, and evidence of greatly increased peristaltic activity with an almost constant expression of thin, ribbonlike, but otherwise normal-appearing bowel movements.

Examination of the urine was negative except for 3 to 5 white cells per high-power field. The blood showed a red-cell count of 4,550,000 and a white-cell count of 43,200, a smear showing 50 per cent young polymorphonuclears with 21 per cent myelocytes. A blood Hinton reaction was negative. Many *Staphylococcus aureus* colonies and a moderate number of beta-hemolytic streptococcus colonies were obtained on throat culture. Stool cultures were negative for typhoid-dysentery organisms.

A roentgenogram of the chest showed a slight increase in the lung markings, interpreted as probably of no significance. Films of the abdomen and sacral spine were not remarkable.

The patient remained in the hospital for nine weeks. She continued to have frequent bowel movements, to show signs of vasomotor instability, to exhibit hyperperistalsis, and to run a persistent tachycardia of 150 to 180. The temperature rose occasionally to around 100°F. An electrocardiogram showed only sinoauricular tachycardia. The urine continued to show occasional pus cells, and on two occasions cultures of it grew out colon bacilli and *Streptococcus faecalis*. The blood picture remained much the same as on entry except that the white-cell count stabilized itself at from 10,000 to 15,000. The stools were occasionally guaiac positive. Repeated stool cultures showed no typhoid-dysentery organisms. A single blood culture was negative.

A gastrointestinal series revealed marked increase in the motility of the stomach and the small

*This case is presented through the courtesy of the Children's Hospital, Boston.

intestine but no specific lesions. A barium enema performed shortly after admission revealed a decrease in the caliber of the colon, which was spastic and irritable, but exhibited an apparently normal mucosal pattern. Intravenous pyelograms showed delay in excretion of the dye but no morphologic abnormalities. Urea clearance was 22 per cent of normal. A lumbar puncture was entirely normal.

The patient required frequent parenteral fluids and was transfused liberally, but was resistant to various forms of treatment. Paregoric and luminal were ineffective, and belladonna was tried once, but resulted in a very severe reaction, with dehydration, fever, peripheral vasomotor collapse, dilatation and fixation of the pupils, and abdominal distention. In the fourth week, sulfadiazine was started but was without beneficial effect. Water and electrolyte balance was maintained with difficulty because of persistent diarrhea, but in spite of all the measures adopted, together with changes of formula, the patient failed to gain in weight. The blood chemical values (calcium, phosphorus, phosphatase, nonprotein nitrogen, carbon dioxide and sodium chloride) remained essentially normal, except for a slight reduction in serum protein.

During the seventh week of hospitalization, the patient developed jaundice, which progressed steadily in severity. The icteric index was 110 two weeks before death, and urobilinogen was absent from the urine. With the increase in the jaundice, the abdomen became increasingly distended and tense, but peristalsis remained as active as ever. During the last few days of life, there was fluoroscopic evidence of enlargement of the liver and spleen, as well as distention and prominence of the veins in the abdominal wall. During the eighth hospital week, the patient became essentially moribund, vomited large amounts of guaiac-positive coffee-grounds material, and suffered a marked weight loss, with dehydration and a subnormal temperature. Terminally, there was fluoroscopic evidence of infiltration of the right upper lobe, but otherwise the chest remained clear. The patient died on the sixty-fourth hospital day.

Differential Diagnosis

Dr. Joseph Garland: It is hard to decide where to begin in a case like this. Ordinarily, when an infant shows some definite departure from normal physiology, we think of the cause first in terms of infection, parenteral or enteric, most commonly the former, and failing this, some other disturbance of the environment. We know how infants react to heat and cold, emotional stimulation, alterations in the type of food ingested, and lastly, we must consider some congenital anomaly or deficiency, or failure of some organ or system to respond normally to the demands of existence. Any of these, but particularly infection, is apt to give rise to gastrointestinal disturbances as one of the predominating symptoms, so that in this baby I think we should first think of infection as causing her sickness and we can find in this record plenty of evidence on which to predicate an infection. For example, on admission to the hospital, a slight pharyngitis was apparently present. There was a definitely abnormal white-cell count, so high as to be quite confusing, and a definitely abnormal differential count—50 per cent young polymorphonuclears and 21 per cent myelocytes. I assume that this abnormality can be accounted for by infection, even an infection of the hidden type and one that did not cause any great thermal response. Normally, at this age, the white-cell count may run as high as 12,000, but rarely over that. Also at this age, a differential count is more apt to show predominance of lymphocytes over the polynuclear series. On account of the relatively normal red-cell count, the leukocytosis was obviously not due to simple anhydremia. Furthermore, 1 per cent of myelocytes is probably normal at this age. Therefore, something definite caused the blood picture to go wrong. Later on, however, we know that the white-cell count stabilized itself at 10,000 to 15,000—that is, from normal to slightly above normal. The blood picture otherwise remained the same as at entry. In other words, we assume that no anemia developed and that the differential count continued to show predominance of the myelocytic series. The positive cultures from the throat may or may not be of significance, but we must consider that they might also point to actual disease.

The urine continued to show occasional pus cells, and it is not stated whether these were catheterized specimens, although I assume that some at least were, and on two occasions colon bacilli and streptococci were grown. There is therefore a possibility of a renal infection, and any parenteral infection may cause severe gastrointestinal symptoms. Accordingly, we have a right and almost an obligation to think first in a case like this of so-called "infectious diarrhea of the newborn," which means ordinarily a parenteral infection of the type that a few years ago was brought out in the literature as the "hidden-mastoid" type of diarrhea because a certain number of cases were found in which there was no obvious respiratory infection but in which infection of the mastoid was present.

This illness continued a very long time, without showing any definite change, and other factors are certainly mystifying. In the first place, although the word "diarrhea" is used in the case history, there is nothing that actually gives us an indication that this baby had diarrhea. It is true

that she had marked hyperperistalsis, with a considerable discharge of feces, but wherever these feces are described they were normal in consistence and normal or close to normal in color, although containing at times fresh blood and at other times showing a positive guaiac reaction. Yet we must remember that there was a rectal prolapse, which might have caused fresh blood. In any type of diarrhea with which I am familiar, whether it is infectious diarrhea or diarrhea due to either environment or food disturbance, I should expect to find looseness of the stools and mucus in the stools. In other words, although this case was called diarrhea, I am not willing to accept it as diarrhea on the evidence presented. What we can say is that there was definite increase of peristalsis, with spasm and pain, and that at other times the baby acted and ate in a normal fashion; this went on for nine weeks before eventual collapse took place.

The other evidences of disturbance are the urine, which showed on a couple of occasions a positive culture and on several occasions a very few white blood cells, a blood picture that was definitely abnormal and may have been the result of infection, the original chest x-ray film that, although it was interpreted as not significant, nevertheless showed some slight increase in the lung markings. Then we have some curious cardiac signs and symptoms — tachycardia, cyanosis and signs of vasomotor instability. These, I think, probably tie in with almost any severe disturbance that an infant of this age might have. If we can accept the diarrhea as a symptomatic diagnosis, I think that acute infectious diarrhea of the newborn is the most probable diagnosis. I am not willing, however, to accept this as being a case of diarrhea, and failing that diagnosis, I think anyone who tries to discuss the case is led into the wilderness of conjectures, of possible anomalies or other types of disease that do not show themselves as a characteristic picture. We have seen lately an occasional case in the newborn of cystic fibrosis of the pancreas, for example, which does not show diarrhea but does show abnormal stools and steatorrhea; one can almost take it for granted that there was a lack of vitamin A absorption and consequently an avitaminosis with abnormalities of the epithelium wherever it existed — severe disturbances with eye changes and usually, I believe, with a definite tendency to upper respiratory infection, even bronchiectasis. I cannot actually fit that into this picture, however.

We also have a number of congenital abnormalities, some of which might fit this picture, and some of which might go along with it. Duodenal ulcer of the newborn is far from being a common condition but does occur, and the diagnosis in life is extremely difficult to make. It is a condition in which hemorrhage is almost the only feature that may be discovered. In fact, the diagnosis of duodenal ulcer is usually made only as the result of hemorrhage. Did an anomaly such as an atypical duodenal ulcer, atypical Meckel's diverticulum or partial intussusception start this off? I cannot get any nearer to it than that. A family history of allergy is mentioned, but I have never seen an infant with food allergy in whom vomiting was not the predominating symptom; in this case, vomiting was almost lacking except for two occasions, and then at the end of the picture. Certainly, there would be a secondary avitaminosis in a case like this in which so little food was apparently being absorbed over such a long period. There was obviously a terminal infection, and at the end of the disease there were jaundice, enlargement of the liver and spleen and, possibly, a hepatitis.

DR. RICHARD SCHATZKI: I have been given four films, two of the small and two of the large intestines, taken approximately two months before death. They show that the liver is approximately of normal size and normal density. The spleen is not clearly visible but appears to be not appreciably enlarged. The small intestine, so far as I know, looks normal for a baby four weeks of age. My own experience with the small intestine of such a patient is not very large, but Dr. S. G. Henderson, of Pittsburgh, at a recent meeting of the American Roentgen Ray Society, presented a series of 100 cases of small intestines in babies during the first week of life that, so far as I remember, looked like this.

DR. ALLAN M. BUTLER: Do you want to remark on the fact that in an older patient a distribution of barium like that indicates certain things whereas in the infant it does not?

DR. SCHATZKI: I think quite a few people would in an older person call this a deficiency disease. There has been some dispute in the literature regarding the appearance of the small intestine in the newborn. Some authors maintain that the newborn normally shows a pattern that in an adult is characteristic of deficiency disease. Dr. Henderson showed a large number of films in which the small intestine of the same patient, a baby, varied from the so-called "deficiency pattern" to that of a normal adult. In other words, he thought the small intestine in the newborn had characteristic folds, which had been denied by others. As I said, I should call this normal small intestine. My knowledge of barium in the colon is still more limited, because I have not done many barium enemas on babies of this age, but I should agree with the report that this colon was too narrow. In addition, it was too short and had no real splenic flexure. The splenic flexure was as low as the hepatic flexure — not because of a large spleen but because the colon was short. The

pattern, I should agree, was probably normal, although I think that the mucosa was a little too thick—the mucosa and submucosa together. I do not know what causes a short and narrow colon in an infant a few weeks after birth. I remember that we had a case not too long ago in which a baby had a narrow colon. Somebody told me that the Children's Hospital had found narrow colons in cases with cystic fibrosis of the pancreas.

DR. TRACY B. MALLORY: Have you anything further to add, Dr. Garland?

DR. GARLAND: I am afraid I do not know anything more.

DR. MALLORY: As I understand it, your preference is for an underlying infection whose nature you do not know.

DR. GARLAND: On the premise of diarrhea, yes; otherwise, no.

DR. BUTLER: I think it is only fair to say that the physicians at the Children's Hospital who took care of this patient were as puzzled as Dr. Garland, even though they recognized the importance of the autonomic disturbance. Had Dr. Garland seen the child, he would have been more impressed by her vasomotor instability and autonomic imbalance. These were very striking and are hardly appreciated in a reading of the few sentences that relate to them in the written presentation of the case.

DR. WYMAN RICHARDSON: Do infants of this age ever have idiopathic ulcerative colitis?

DR. GARLAND: This patient was very young for it.

CLINICAL DIAGNOSES

Chronic nutritional disturbance.
Pneumonia.
Hepatitis, acute, infectious.

DR. GARLAND'S DIAGNOSES

Hyperperistalsis, cause unknown.
Terminal infection, with hepatitis.

ANATOMICAL DIAGNOSES

Idiopathic ulcerative colitis, with perforation and peritonitis.
Bronchopneumonia, terminal.
Hepatitis and pyelonephritis, mild.
Pancreatic fibrosis (achylia), early.

PATHOLOGICAL DISCUSSION

DR. SIDNEY FARBER*: Not only the physicians who saw this patient but also the pathologists were disturbed. The main clinical working diagnosis was autonomic imbalance. The final discharge diagnosis was chronic nutritional disturbance, underlying etiology undetermined, pneumonia and acute infectious hepatitis. There was a bronchopneumonia, particularly of the left upper lobe, but this was of recent origin.

This is the most important finding: in the region of the transverse colon, there was localized peritonitis, some of which was well organized. In addition, there was diffuse peritonitis from which colon bacilli were obtained. The transverse colon proved to be the site of the previous perforation of the bowel. When the bowel was opened, large numbers of ulcers were found involving the transverse colon, descending colon, most markedly, and also the ascending colon and the rectum, less markedly, however. The involvement of the bowel stopped rather abruptly at the ileocecal valve. The gross microscopic picture was characteristic of ulcerative colitis. There was also a diffuse hepatitis, with a considerable amount of inspissation of bile in the biliary canaliculi. Most of the icterus noted was presumably of the obstructive type. There was a very early lesion of the pancreas acini typical of the picture associated with achylia pancreatica. The mucous glands of the trachea and bronchi and some of the glandular structures of the duodenum showed inspissation of mucus and dilatation of the glandular structures. There was infection, a colon bacillus bacteremia. Whatever organisms might have been there before were certainly obscured by this terminal infection. There was some evidence of older infection in the upper respiratory tract, otitis media and slight, healed pyelonephritis. Whatever the cause of this disease may have been, we have not been able to discover it in this case, although we had an ideal opportunity here, since the symptoms began so early after birth. If one compares this picture with a disease somewhat like it, mucous colitis, and if one recalls the stress that Drs. White, Cobb and Jones† and their colleagues have placed on the probable role of the nervous system, a similar direction may be followed here. The tachycardia, the vasomotor instability and the gastrointestinal disturbance may all be grouped together etiologically from that point of view.

I should like to ask Dr. Mallory what he now considers the cause of ulcerative colitis.

DR. MALLORY: If I were to make a guess, it would be along the same general lines—very definitely against a specific infection.

DR. SCHATZKI: How common is this? Is it very rare in babies?

DR. FARBER: In young infants of this age, yes. We have had two under six months in my experience; this one at three months, and one at two.

DR. BUTLER: Did the one at two display this imbalance?

DR. FARBER: Not to such an extent, but in going over the record, one could find evidence of it.

*Assistant professor of pathology, Harvard Medical School; pathologist, Children's Hospital, Boston.

†White, B. V., Cobb, S., and Jones, C. M. *Mucous Colitis: A psychological medical study of sixty cases.* Psychosomatic Medicine Monograph I. 103 pp. Washington: National Research Council, 1939.

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MASSACHUSETTS LICENSES GENERAL HOSPITALS

Under the provisions of Chapter 661 of the Acts of 1941, the Massachusetts Department of Public Health has promulgated rules and regulations for the licensing of general hospitals and sanatoriums in Massachusetts, which appear elsewhere in this issue of the *Journal*. Although only two other states, Connecticut and Minnesota, have accepted the licensing of general hospitals as a public function, the principle is by no means new in Massachusetts. As early as 1867, the State Department of Public Welfare was authorized to issue a "license to any person it deems suitable to establish and keep a maternity hospital or ward or other place for the reception, care and treatment of women during pregnancy or delivery." This action was followed in 1909 by the licensing by the Department of Mental Health of private hospitals for patients with mental diseases.

In spite of this recognition of the public interest in hospitals and in the standards of hospital care, it has, up to the present time, been possible for any person, lay or medical, to establish and maintain a general hospital anywhere in Massachusetts if his application was approved by the selectmen or board of aldermen of the town or city in question. The voluntary supervision of hospitals that has been carried on for many years by the American College of Surgeons and the American Medical Association has been of the greatest value in bringing the standards of hospital care to their present high level but is, of course, effective only in the institutions that choose to avail themselves of it. The Massachusetts Medical Society and the Massachusetts Hospital Association, by their support of this bill, have expressed the opinion that the citizens of Massachusetts are entitled to the assurance that all hospitals, on which they depend for the most vital part of their medical care, meet the generally accepted professional standards of service.

It is not the object of the licensing act or the intention of the regulations adopted thereunder to tell the physicians of Massachusetts what they may or may not do in hospitals but rather to ensure that the practice of medicine in hospitals is carried on in suitable quarters with the aid of properly trained assistants, with adequate equipment and with such laboratory and other auxiliary services as are recognized as essential to modern hospital care. For the further protection of patients, the hospital records must be sufficiently complete to make clear the diagnosis and to show how it is established, and to describe concisely the treatment given, including the type of operation and operative findings. The value of such records to physician and patient alike is universally recognized in professional circles, and adequate medical records have been a major factor in the improve-

ment of hospital services during the past fifty years.

The detailed requirements for maternity hospitals and nurseries may seem unnecessarily exacting to the generation of practitioners who have been accustomed to think of obstetrics as a routine part of general practice. The Massachusetts standards, however, are substantially less exacting than the widely commended maternity-hospital regulations of New York City and of Chicago. Although recognizing the limitations of small hospitals in meeting optimum modern requirements, the new state regulations definitely reflect the recent attempts of the Massachusetts Medical Society and the Department of Public Health to throw every reasonable safeguard around childbirth and further to reduce the excessive maternal mortality rate.

This assumption of responsibility for minimal standards of hospital service by an official agency does not constitute "state medicine." Under the provisions of this act and the adopted regulations, all licensed physicians are free to practise in hospitals in accordance with the accepted standards of their own professional organizations. Hospital licensing on this basis is simply a recognition of the citizens' interest in what has become an essential public service.

MEDICAL BIBLIOGRAPHY OF SIXTEENTH-CENTURY AUTHORS

Soon after printing began, five hundred years ago, interest was shown in books by medical authors. So far as is known, the first book printed of an exclusively medical nature was published in 1471. In the next ten years, however, over two hundred such books appeared, as listed by Osler in *Incunabula Medica* (Oxford, 1923). Many collections of these items from the early presses are known, the most extensive in America being at the Boston Medical Library. Each book in this section of medical literature has been studied meticulously by various scholars, and even with works issued without title page, date, place or printer, experts such as Dr. Arnold C. Klebs are able to supply most of the missing data. It seems probable that no group of objects ever devised by man has received so much attention from the learned world. The work still goes on: Klebs's standard short title list, *Incunabula Scientifica et Medica* (Bruges, 1938), is being constantly checked and even augmented; and Miss Margaret B. Stillwell, of Brown University, has only recently completed *Incunabula in American Libraries: A second census of fifteenth-century books owned in the United States, Mexico and Canada* (New York, 1940).

In view of all the work that has been done on fifteenth-century books, it is surprising how little attention has been paid to those published during the sixteenth century. Most libraries, for instance, have isolated their incunabula, but leave the sixteenth-century books in the stacks, sometimes not realizing that these are as precious and rare as many published in the preceding century. Gradual interest in this group of books, and in the larger list of all items published by persons living in the sixteenth century, has resulted in the issue of the first fasciculus of *Bio-bibliography of XVI. Century Medical Authors* (Washington, 1941), by Claudius F. Mayer, the learned editor of the *Index-Catalogue of the Surgeon General's Library*.

In Dr. Mayer's list will be found many unusual features. Its accuracy and completeness, based on the great collections in the Army Medical Library, are assured. In addition to the usual features in a bibliographic list, one will find the location of known copies of individual books, not only in this country, but abroad. Doubtful or nonexistent copies are referred to, and in most cases a brief biographic notice is given of the author, with a reproduction of his portrait.

This project, now only part way through the letter "A," is a welcome addition to bio-bibliographic reference, particularly so in view of the fact that copies of books known to exist in America are referred to, and the essential facts regarding the authors are given. No one is better equipped than Dr. Mayer to carry out this long-needed work,

and he is to be congratulated on the completion of the first section. Plans have been made to issue the fasciculi regularly; the entire work will take several years.

MEDICAL EPONYM

Pick's Disease

Dr. Friedel Pick (1867–1926), first assistant in the First German Medical Clinic at Prague, presented a discussion, "Über chronische, unter dem Bilde der Lebercirrhose verlaufende Pericarditis (pericarditische Pseudolebercirrhose) nebst Bemerkungen über die Zuckergussleber (Curschmann) [Chronic Pericarditis — Pericarditic Pseudocirrhosis of the Liver — Simulating the Course of Cirrhosis of the Liver, with Observations on the Frosted Liver (Curschmann)]," in the *Zeitschrift für klinische Medicin* (**29**:385-410, 1896). A portion of the translation follows:

> There is a symptom complex (pericarditic pseudocirrhosis of the liver) that simulates one of the mixed forms of cirrhosis of the liver (enlarged liver, marked ascites without jaundice). This results from disturbances in the circulation of the liver caused by a latent pericarditis. These lead to an overgrowth of connective tissue, which produces marked ascites by causing congestion in the portal circulation.
>
> The condition usually occurs in young persons, but may be seen in later life.
>
> The following points are to be considered in the differential diagnosis: absence of any etiologic agent for cirrhosis of the liver, a history indicating a previous pericarditis and a previous edema of the legs. Only careful examination of the heart can finally establish the diagnosis.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

COMMITTEE ON MATERNAL WELFARE

Case History: Fatal eclampsia and cerebral hemorrhage

A twenty-three-year-old primipara was first seen during the seventh month of pregnancy, when she had a blood pressure of 160 systolic, 94 diastolic. Her care from this time on was adequate if not intelligent: she was hospitalized for the remaining weeks of pregnancy. Physical examination on entry was essentially negative, and the past history showed no serious illnesses or operations. Albuminuria did not appear until the thirty-fourth week. The usual pre-eclamptic and toxemic treatment was instituted. The patient did not go into labor until three weeks after the expected date, and the systolic blood pressure ranged from 150 to 160 for the last two weeks, when albuminuria was also constant. After four and a half hours of spontaneous labor, she developed eclampsia accompanied by a cerebral hemorrhage from which she died very shortly. A post-mortem cesarean section was performed, but the baby did not live. Autopsy revealed massive cerebral hemorrhage, with signs typical of eclampsia in the liver and kidneys.

Comment. There is no excuse for this fatality. Any patient under constant observation for two months who runs a constantly elevated blood pressure and albuminuria should not develop eclampsia and die of cerebral hemorrhage. Although the systolic blood pressure is not recorded as having risen above 160, it is very likely that it did, since it is extremely uncommon for a patient with a systolic blood pressure no higher than this to have a convulsion and fatal cerebral hemorrhage. Patients with toxemia who show a systolic blood pressure of over 150 constantly accompanied by albuminuria when they are in the eighth month of pregnancy, and do not improve with treatment become problems for induction, since kidney damage is undoubtedly progressing. A sharp rise in blood pressure or a diminution in the urinary output calls for immediate delivery. If the cervix is unfavorable for induction, cesarean section is the operation of choice. Depending on the condition of the cervix, either one or the other should have been performed at least two weeks before this patient died.

Post-mortem cesarean sections are a moot question. In this case, as so often happens, nothing was accomplished. It is very gratifying that an autopsy was performed. It tells the true cause of the fatality.

DEATHS

BUTLER — Francis J. Butler, M.D., of Worcester, died June 7. He was in his sixtieth year.

Dr. Butler received his degree from Georgetown University School of Medicine in 1908. He was a member of the staffs of St. Vincent and Worcester Hahnemann hospitals, and was a fellow of the Massachusetts Medical Society and the American Medical Association.

COX — Stanley C. Cox, M.D., of Holyoke, died June 7. He was in his sixtieth year.

Born in South Hadley Falls, Dr. Cox received his degree from University of Michigan Medical School in 1910. For twenty-one years, he served as associate medical examiner and medical examiner for the third district of Hampden County. He was head of the local Medical Division of the Massachusetts Committee on Public Safety in Holyoke. He was a member of the American College of Physicians and Surgeons, and a fellow of the Massachusetts Medical Society and the American Medical Association.

His widow, two sons, three daughters, two brothers and a sister survive him.

WILLIAMS — DAVID L. WILLIAMS, M.D., of Winthrop, died June 7. He was in his sixty-seventh year.

Born in South Boston, Dr. Williams received his degree from Tufts College Medical School in 1906. He was a former member of the staffs of Harvard and Tufts College medical schools. From 1936 to 1938, he was state commissioner of mental diseases. He was a former member of the Massachusetts Medical Society and the American Medical Association.

His widow, two sons, two daughters and a brother survive him.

WAR ACTIVITIES

CIVILIAN DEFENSE

CLARIFICATION OF FUNCTION OF RED CROSS AND CIVILIAN DEFENSE UNITS

The following joint statement concerning the respective authority and responsibilities of the local Red Cross chapters and the local Civilian Defense councils was issued on May 18, 1942:

To secure unity of effort and avoid duplication of facilities in meeting civilian needs arising from enemy action, this statement is issued by the Office of Civilian Defense and the American National Red Cross for the guidance of defense councils and Red Cross chapters.

It is the responsibility of local defense councils to see that adequate provision is made for all services required in the event of bombing or other enemy attack. During an emergency period, the commander of the Citizens' Defense Corps will exercise control over all such services.

With respect to emergency medical services and emergency feeding, housing and clothing, provision should be made in each community in conformity with the following principles:

EMERGENCY MEDICAL SERVICES

During bombing or other enemy attack, all services are directed from the control center in charge of the commander of the Citizens' Defense Corps. Responsibility for the care of those injured as a result of enemy action rests with the Emergency Medical Service of the Citizens' Defense Corps under the direction of the chief of the Emergency Medical Service.

Red Cross chapters assist the Emergency Medical Service by (*a*) recruiting and training volunteer nurses' aides, who will be utilized by the Emergency Medical Service at base and casualty hospitals, casualty stations and first-aid posts; (*b*) furnishing lists of persons trained in first aid to be enlisted by the Emergency Medical Service as members of its stretcher teams; (*c*) providing dressings, bandages and supplementary equipment as the chapter may decide in consultation with the chief of Emergency Medical Service; (*d*) equipping and operating emergency ambulances to be assigned to the Emergency Medical Service and to serve under its direction; (*e*) providing supplementary transportation for walking injured and for Emergency Medical Service personnel. During the emergency period, ambulances and motor units assigned to such transportation service will be under the direction of the chief of Emergency Medical Service or the transport officer. The Emergency Medical Service of the Office of Civilian Defense will not be duplicated by the Red Cross but will be utilized by the Red Cross in natural disasters.

EMERGENCY FEEDING, HOUSING AND CLOTHING

In the joint statement, dated April 17, 1942, of the Office of Defense Health and Welfare Services and the American Red Cross it is agreed: "With respect to the emergency period during which special facilities must be made available to meet emergency needs without notice, the Federal Security Administrator will look to the local facilities and resources of the American Red Cross to provide food, clothing and temporary shelter." These services will be provided locally during an emergency period by the Red Cross under the control of the commander of the Citizens' Defense Corps in accordance with detailed plans to be worked out jointly by the commander, the Red Cross chapter and the public-welfare authority.

Defense councils should avoid duplication of these facilities. Where an emergency food and housing corps has already been organized and equipped to the satisfaction of the commander of the Citizens' Defense Corps, its function should be co-ordinated with the functions of the public-welfare authorities and the Red Cross chapter and, if possible, consolidated.

After the emergency period, the appropriate public agencies are expected to undertake the care of civilians in accordance with plans developed in conjunction with the Office of Defense Health and Welfare Services and the Federal Security Administrator. Funds will be made available for this purpose by the federal government through the Federal Security Administrator. Local welfare agencies and Red Cross chapters should be guided in their relations by the agreement signed on April 17, 1942, by the Office of Defense Health and Welfare Service and the American Red Cross.

All Red Cross volunteers enlisted in the emergency feeding and housing service, and all other Red Cross volunteers who are to be in service during and following bombing or other enemy action, will register with the local Civilian Defense volunteer office. The cards of all such registrants are to be marked so as to show that these volunteers are in Red Cross service. In order to obtain necessary freedom of movement during and immediately after enemy action, Red Cross personnel certified to the commander by the chapter for emergency feeding, housing and clothing services will be furnished with identification cards issued to Citizens' Defense Corps personnel and will be authorized to wear the official armband.

This statement supersedes the joint statements of September 4 and December 22, 1941.

NORMAN H. DAVIS, *Chairman*
American Red Cross

JAMES M. LANDIS, *Director*
Office of Civilian Defense

MISCELLANY

PROMIN IN THE TREATMENT OF TUBERCULOSIS

Success in the use of chemotherapeutic agents in combating infectious diseases revived the hope that eventually a substance will be found that will be useful clinically in the treatment of tuberculosis. Promin, one of the compounds used experimentally, has already been discussed

in the public press. A recent article (Feldman, W. H., Hinshaw, H. C., and Moses, H. E. Promin in experimental tuberculosis. *Am. Rev. Tuberc.* **45**:303–333, 1942) announces the results obtained in animals with this chemical.

In 1938, sulfanilamide was reported to have an inhibitory effect on the development of experimental tuberculosis in guinea pigs, but subsequent papers held out scant hope that this agent would prove to be a specific remedy for tuberculosis in human beings.

Promin, in its solid form, varies from white to light yellow and is slightly hygroscopic. It is highly soluble in water, and 40 per cent solutions are stable indefinitely and may be sterilized by heat. It is slightly bitter, but small amounts may be mixed with the food of animals without impairing their appetite or digestion. Guinea pigs tolerate 1 per cent promin in their foods and will consume from 300 to 400 mg. of the drug per twenty-four hours. Increasing the concentration of promin to 2 per cent causes anorexia, which interferes with the quantity of food taken.

In the first experiment, promin to the amount of 1 per cent was added to the food of 30 guinea pigs; 20 others received the same diet except for promin. Two days after the feeding experiment began, all guinea pigs were inoculated subcutaneously with human tubercle bacilli of known strain. On the eighty-fourth day, all the animals in the control group had died, and 24 of the animals that had received promin were living. Promin was then removed from the diet of 12 of the survivors. After eighty-two more days, 13 animals still lived, 5 of which had received promin for the entire period (one hundred and sixty-six days) and the other 8 for the first period of eighty-four days only. The purpose of this procedure was to determine if latent tuberculosis would become reactivated when treatment was discontinued.

The value of a chemotherapeutic agent must be appraised not only on survival time, but also on the character of the disease process. With one exception, the degree of tuberculous involvement in the animals that received promin was notably less than that in the controls.

Although the results indicated that in many of the animals promin had either prevented the establishment of lesions or caused their eventual disappearance, another effect of the drug, which is perhaps of more importance, was that exerted on the cellular elements of the lesions. In the vast majority of the animals in the treated group that had lesions, the histologic characteristics of the pathologic process apparently were modified favorably. This was especially true of the lesions in the parenchymal tissues. The lesions were usually small and discrete, and the epithelioid phase of the reactive process predominated. Necrosis was infrequent, and a tendency of the process toward fibrosis was frequently observed. These features of the morbid process were in marked contrast to those that characterized the disease in the control group of animals. In the latter, the disease was extensive, destructive and progressive.

The objectives of the second experiment were to confirm the results of the first and to determine what effect, if any, promin might have on a tuberculous infection introduced at the same time as or at varying periods before treatment with promin was begun.

Eighty guinea pigs were selected and divided into eight groups. Group I consisted of 12 animals infected but not treated (controls). Group VIII consisted of 20 animals whose treatment began two days prior to infection. Groups II to VII each contained 8 animals, and treatment was begun, in relation to the day of infection, at various intervals as follows: Group II, on the day of infection; Group III, after three days; Group IV, after one week; Group V, after two weeks; Group VI, after four weeks; and Group VII, after six weeks.

All animals except 1 reacted to tuberculin. Generally speaking, the reactions of the animals that received promin were less severe than those of the untreated animals.

Although the general physical condition of the animals remained satisfactory, changes indicative of toxic manifestations were noted in the blood and spleen. Studies, as yet incomplete, indicate that in guinea pigs, promin may induce a hemolytic type of anemia but with adequate regeneration, as indicated by a corresponding reticulocytosis.

The difference in survival times of the several groups was striking. When the last of the untreated animals died, one hundred and eighty-nine days after inoculation, 84 per cent of the treated animals were still living. Of the treated animals that died, none had sufficient tuberculosis to account for death, and this percentage of deaths might reasonably be considered an average or normal mortality rate for guinea pigs.

Examination of the tissues and organs of the animals showed that all untreated animals were tuberculous, that in 57 per cent of the treated animals no evidence of infection in the visceral organs was found, that, in the remainder of those treated, tuberculosis was found (with a few exceptions) of minimal severity, and that 43 per cent of the treated animals failed to show evidence of disseminated tuberculosis.

The failure to demonstrate lesions of tuberculosis in a considerable number of the animals that were treated and the fact that the disease in the treated animals was, with few exceptions, minimal and nonprogressive indicate that the action of the drug was significant. That fairly comparable results occurred in the treated animals, regardless of whether the administration of the drug was started before or as long as four or six weeks after inoculation with tubercle bacilli, was surprising and must indicate that the drug was effectively operative against a tuberculous infection in which morbid changes already were established when administration of the drug was started.

The conclusion of the two experiments is that promin has a deterrent effect on experimental tuberculous infection.

Encouraged by these carefully controlled animal experiments, workers have cautiously used promin in the treatment of a few cases of tuberculosis in human beings. Administration of the drug has proved difficult, since its toxic effect in man is found to be much higher than that in the guinea pig. In certain cases, it has been found necessary to discontinue treatment because of unfavorable symptoms attributed to the drug itself. In other cases, in which treatment has been prolonged (five months or more), results thus far show varying effects. In a few, definitely demonstrable improvement occurs; in others, little or no change is observed. In some patients, the disease goes on developing progressively, with no apparent effect from the treatment.

It is obvious that a freshly infected guinea pig presents a very different pathologic picture from that of a well-developed human case with destruction of tissue and extensive fibrosis interfering with access of the drug to living tubercle bacilli.

Despite the present lack of convincing evidence of promin's value in the treatment of human tuberculosis, there is a definite belief that further trial in skilled hands is indicated. — Reprinted from *Tuberculosis Abstracts*, June, 1942.

THE BEGG SOCIETY

A medical-discussion group, to be known as The Begg Society, has been formed at Boston University School of Medicine in honor of the late Dr. Alexander S. Begg, dean of the school for seventeen years. An annual prize will be offered to the student author of the best scientific paper submitted by a member of the society. Dr. Chester S. Keefer was elected president by the students, and Drs. Joseph F. Ross and Francis M. Forster were named to the prize committee.

NOTE

At the recent annual meeting of the American Gastro-Enterological Association, held in Atlantic City, Dr. Sara M. Jordan, of Boston, was elected president for the ensuing year.

CORRESPONDENCE

REFUGEE PHYSICIANS

To the Editor: The Greater Lawrence Medical Association has always been able to handle its local problems, but now we are confronted with a situation that is statewide; and we appeal to the Massachusetts Medical Society for its solution.

Massachusetts has had the reputation for being a "dumping ground" for physicians not wanted elsewhere; once again we have that undesirable distinction, as evidenced by the influx of alien physicians in this state. No doubt the Board of Registration in Medicine is being swamped with their applications.

All the eligible members of our organization have volunteered their services either to the armed forces or to federal services through the Procurement and Assignment Forms. We are ready to sacrifice everything for our country; yet, is it Americanism to penalize us with the thought that our patients, whom we have served with the best of our ability with honest medicine, should suddenly be left to the mercy of ignorant and incompetent alien doctors?

There are over 200 refugee doctors practicing in Massachusetts; and there will be many, many more unless something is done quickly. Recently, we raised the standards of certain unrecognized schools; yet we allow these refugee physicians in Massachusetts, whose class of school and educational background cannot be verified by the examining board.

Major Seeley does not want these men to treat our boys in the army; yet, they are allowed to go into private practice in this hour of trial, when it is essential that the morale of our people be kept at the highest level possible.

Twenty-eight States, either by statute or by a ruling of the examining board, require full citizenship for medical licensure. The Greater Lawrence Medical Association believes that this requisition should be put into immediate effect by the State Board of Registration in Medicine. Within the five-year period required for full citizenship, the alien physician can acquire our ideals, our ethics, and the American way of practicing medicine. Most important of all, this would leave no loopholes for spies, saboteurs or subversive activities. Is this expecting too much from anyone who found existence in another land unbearable? Let us not forget that we are a nation at war.

PHILIP E. ZANFAGNA, *President*
Greater Lawrence Medical Association
126 Haverhill Street
Lawrence, Massachusetts

RULES AND REGULATIONS FOR LICENSING HOSPITALS

HOSPITAL STANDARDS

To the Editor: The following are the rules and regulations established under the authority of Chapter 111, Sections 71 to 73 inclusive (Tercentenary Edition), as amended by Chapter 661, Acts of 1941, for the licensing of hospitals and sanatoriums by the Department of Public Health.

These rules and regulations were approved and adopted at a meeting of the Department of Public Health on April 14, 1942.

PAUL J. JAKMAUH, M.D.
Commissioner of Public Health.
State House
Boston

* * *

RULES AND REGULATIONS FOR THE LICENSING OF HOSPITALS BY THE MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

I. INTRODUCTION

A. *Legal authority.*

1. Chapter 111 of the General Laws (Tercentenary Edition) as amended by Chapter 661 of the Acts of 1941 authorizes the Department of Public Health to issue for a term of two years a license, subject to revocation by it for cause, to any person whom it deems suitable and responsible to establish or maintain a hospital or sanatorium which meets the requirements of the department established in accordance with its rules and regulations. This act also provides for the transfer of licensing of maternity hospitals and maternity wards from the Department of Public Welfare to the Department of Public Health.
2. As a guide in carrying out this responsibility the department, in accordance with authority granted in Section 72 of the above Act, has adopted the following rules and regulations.

B. *Definitions.*

1. Under the provisions of this act a hospital or sanatorium is defined as any institution, whether conducted for charity or for profit, which is advertised, announced or maintained for the expressed or implied purpose of caring for persons admitted thereto for purposes of diagnosis or medical or surgical treatment which is rendered within said institution.
2. The term "maternity hospital" shall mean any hospital institution or place other than the home of a near relative in which pregnant women are cared for or delivered; the term "newborn nursery" shall mean any room, rooms or ward in such hospital, institution or place in which newborn babies are cared for or are treated.
3. The term "department" as used in these regulations shall mean the Massachusetts Department of Public Health.
4. Institutions not included in the provisions of the act are those caring for cases of mental diseases and licensed by the Department of Mental Health, including nervous diseases. It does not include convalescent homes or homes for the aged, dispensaries or clinics.

C. *Issuance of license.*

1. Application for a hospital license must be made in writing upon a form provided by the department. It shall contain the name of the applicant or if an association or corporation the names of any two officers thereof; the name of the hospital; the total bed capacity of the hospital, and in Groups A and B, the number of beds for patients on each floor; the type of hospital to be operated; the location thereof and the name of the person in charge. The license fee of $10.00, payable to the Commonwealth of Massachusetts, must accompany the application.
2. The applicant must be a person of reputable character and shall provide references satisfactory to the department.
3. Every hospital shall be designated a permanent and distinctive name which shall appear on the application for license, and which shall not be changed without first notifying the department.
4. Applications for a license to establish or maintain a hospital or sanatorium shall be considered by the department only if the local board of health has first certified to the department that, from its inspection and examination of said proposed hospital or sanatorium, it is suitable for the purpose.
5. The license must be conspicuously posted on the premises.

D. *Revocation of license.* A hospital license may be revoked by the department on any of the following grounds, subject to Section 1, Chapter 661, of the Acts of 1941:

1. Violation of the provisions of the licensing act or of the standards, rules or regulations of the department adopted thereunder.
2. Permitting, aiding or abetting the commission of any illegal act in such institution.

Each license shall be returned immediately to the department on its expiration or revocation.

E. *Classification of institutions.* For the purpose of these regulations, all institutions licensed under the provisions of this act are classified as follows:

1. General hospitals with maternity service.
2. General hospitals without maternity service.
3. Maternity hospitals.
4. Tuberculosis hospitals.
5. Other types of hospital as designated.

Hospitals as above classified are divided into the following groups: Group A, 1 to 49 beds; Group B, 50 to 149 beds; Group C, 150 or more beds.

II. General Regulations.

A. *Fire protection.* Provisions for fire protection and egress shall be satisfactory to the hospital supervisor of the department and to an inspector of the Massachusetts Department of Public Safety.

B. *Sanitation.* No system of water supply, sewerage or garbage disposal shall be installed, nor shall such existing system be materially altered or extended, until complete plans and specifications have been submitted to and approved by the department. Any existing system of water supply, sewerage or refuse disposal must meet the requirements for hospitals as established by the department. All construction of such systems must be carried out in accordance with the plans approved by the department.

1. Water supply. The water supply must be of safe, sanitary quality and obtained from a water-supply system approved by the department. Running hot and cold water shall be available on each floor of the hospital.
2. Sewage disposal. Sewage shall be discharged into a municipal sewerage system where such a system is available; otherwise, the sewage shall be collected and disposed of in a manner approved by the department.
3. Plumbing. Toilet facilities shall be provided in accordance with the size and number of beds of the hospital. Plumbing, drainage and other arrangements for disposal of excreta, and infectious discharges and institutional wastes shall be in accordance with the standards established by Chapter 302, Acts of 1938, for plumbing in buildings owned and used by the Commonwealth.
4. Waste disposal. All organic waste matter resulting from surgical or medical treatment shall be disposed of by incineration. All other refuse shall be stored and removed from the premises in a manner which does not create a nuisance.
5. Garbage disposal. Suitable facilities shall be provided for collection and disposal of garbage at frequent intervals in a manner which does not create a nuisance.
6. Screens. All outside doors, windows and other outside openings shall be screened against flies and mosquitoes, and proper protection provided against vermin.

C. *Heating.* Heating plant shall be adequate to maintain a cold weather temperature of 70°F. in all rooms used by patients.

D. *Lighting.*

1. Each patient's room should be an outside room with a satisfactory amount of natural light. The window area shall be not less than one eighth of the floor area. All service rooms and working centers shall be adequately lighted. Hallways not otherwise satisfactorily illuminated shall be provided with artificial light giving sufficient illumination to visualize clearly stairways and exits of said halls, night and day.
2. All permanent electric installation shall comply with the National Electric Code of the National Board of Fire Underwriters and state and local building regulations.
3. Emergency lighting facilities must be provided for operating suites and emergency rooms, as a storage battery system or battery operated lamps.

E. *Ventilation.* Buildings shall be at all times adequately ventilated. Kitchen, bathrooms and service rooms shall be so located and ventilated as to prevent offensive odors from entering patients' rooms and public hallways.

F. *Kitchen and food handling.* The kitchen, storeroom and other rooms where food or materials used in the preparation of food are stored, exposed or

handled in the process of preparation or consumption shall be in a clean and sanitary condition, and protected from contamination of any kind. Kitchen facilities shall include refrigeration, and perishable goods must be kept at a temperature low enough to prevent deterioration.

G. *Laundry.* The institution shall be equipped with the necessary laundry facilities for the proper cleansing of linen and other washable goods, or satisfactory arrangement be made for such cleansing in a commercial laundry.

H. *Rooms and wards.*

1. Except for nurseries and children's wards, rooms shall be of sufficient size to allow not less than 50 square feet of floor space per bed, and at least 3 feet between beds.
2. Screens shall be provided in wards or multiple bedrooms in order to secure privacy for each patient. Permanent bed curtains are desirable.

I. *Equipment and furnishings.*

1. There shall be satisfactory provision for the care of clothing, toilet articles and other personal belongings of the patients.
2. A sufficient supply of clean bedding, bed linen, towels and other supplies shall be available for use at all times.
3. There shall be sufficient equipment for nursing care according to type of patients accepted by the institution.
4. Means for signaling nurses must be provided within easy reach of patients confined to bed.
5. Adequate facilities shall be provided for hand washing for personnel.
6. Proper sterilizing equipment shall be provided in accordance with the needs of the type of patients treated.
7. Adequate toilet and bathing facilities shall be provided on each floor in accordance with the standards of the department.
8. There shall be a utility sink on each floor of the hospital.
9. There shall be satisfactory facilities in the utility room for the disposal of contents of the urinals and bed pans, and for cleansing of the same.

J. *Laboratory facilities.* Hospitals in Group A shall provide laboratory facilities for performing routine chemical and microscopic examinations of the urine and complete blood counts. Such clinical laboratory facilities should be under supervision of an experienced clinical pathologist. Such hospitals shall also regularly utilize the services of an experienced clinical pathologist or an approved laboratory for complete clinical, hematological, bacteriological and chemical examinations, as indicated.

Hospitals in Groups B and C shall have a laboratory capable of making the standard hematological, bacteriological, pathological and chemical diagnostic examinations under the supervision of a competent pathologist, or have regular arrangements for obtaining all such services from an approved laboratory.

All abnormal tissues removed at operation, except tonsils and adenoids, shall be examined by the pathologist and a report thereon entered in the patient's record.

K. *X-ray.*

1. Hospitals in Group A which maintain a medical or surgical service shall provide at least a shockproof portable x-ray machine under the supervision of a competent medical roentgenologist.
2. Hospitals in Groups B and C shall have x-ray equipment capable of performing standard diagnostic roentgenological examinations, such as gastrointestinal series, gall-bladder examinations, chest films and so forth, under the direction of a competent medical roentgenologist. A fluoroscopic unit shall be included.

L. *Drugs.* Drugs, poisons, stimulants and medicines shall be plainly labeled and securely locked in a cabinet, closet or separate room, accessible only to the physician or nurse in charge.

M. *Personnel.*

1. There shall be a competent, well-trained executive officer or administrator with authority and responsibility to carry out the policies of the hospital as authorized by the governing board or owner.
2. There shall be a staff of physicians and surgeons licensed to practice medicine in the Commonwealth of Massachusetts. This staff shall be organized with a chief of staff or medical director. Hospitals of Groups B and C shall be so organized that there shall be a responsible head of each department. There shall be an advisory group of recognized consultants in the fields of medicine covered by the hospital.
3. There shall be a set of by-laws, rules and regulations which are approved by the medical staff, signed by the chairman and secretary and approved by the governing board or owner.
4. There shall be a superintendent of nurses who is a registered nurse, and there shall be at all times at least one registered nurse on duty.
5. The operating room and delivery room shall be under the supervision of registered nurses.
6. The ratio of nursing staff to patients shall be adequate for satisfactory care of the patients.
7. All hospitals shall have a registered physician available on call at all times. (On account of war conditions it is not possible to set up a definite requirement in regard to resident physicians at this time.)

N. *Medical records.*

1. The hospital shall keep complete and accurate records of each patient from the time of admission to the time of discharge. Such patient records shall include: adequate identification data; family, personal, past and present illness history; physical examinations; reports of special examinations, including reports of clinical and pathological laboratory findings and x-ray findings; records of consultations; provisional diagnosis; reports of anesthesia and surgical operations performed; treatment notes and progress notes; temperature, pulse and respiration charts; final diagnosis and discharge note, signed by the physician.
2. In case of autopsy, a copy of the autopsy protocol shall be attached to the record.
3. All notes shall be legibly written or typed and signed.

4. A complete list of all births and deaths occurring in the institution shall be sent to the clerk of the town in which the hospital is located, within the first five days of the following month, in accordance with the provisions of Section 6, Chapter 46, of the General Laws.
5. A summary annual report of the activities of the hospital on forms provided by the department shall be filed with the department within three months of termination of each calendar year.
6. The following additional records shall be kept in a form and manner prescribed by or acceptable to the department: record of admissions; register of deaths; daily census record; clinical records; narcotic registers of records; records of physicians' orders; autopsy protocols.

III. Regulations for Surgical Department.

In addition to the general regulations, all hospitals in which surgical operations other than emergency operations are performed shall conform to the following regulations:

A. *Operating room.* The operating room shall have impervious floor and washable walls. There shall be a satisfactory means of illumination of the operative field as well as general illumination. There shall be facilities for scrubbing, preferably in a separate room, with running hot and cold water delivered through spray faucets and controllable without use of the hands. Heating shall be adequate to maintain a temperature of 80°F. in the operating room. A suction apparatus shall be available.

B. *Surgical equipment.* The hospital shall constantly have on hand sufficient instruments to perform an abdominal operation and a supply of sterile dressings and linen. There shall be an operating table so built that it can easily be placed in lithotomy or in Trendelenburg position with the patient on the table.

C. *Sterilization.* The equipment shall include an autoclave capable of operation under 15 pounds steam pressure and a boiling water sterilizer. An acceptable aseptic technic shall be observed in all major or minor operative procedures. Proper care shall be taken to prevent contamination of the surgical field, sterile tables or operating team by visitors. Likewise, after an operation on a septic case, the operating room shall be thoroughly cleansed in a manner adequate for the type of contamination existing.

D. *Anesthesia.* There shall be satisfactory equipment to allow the administration of nitrous oxide, oxygen and carbon dioxide. In operating rooms using anesthetic gases capable of exploding under certain conditions of concentration, humidity and so forth, all reasonable precautions shall be taken to avoid this hazard.

E. *Staff.* Surgical operations shall be done only by physicians designated by the hospital as qualified to do surgery.

F. *Preoperative care.* Operations under a general anesthetic (inhalation, spinal, intravenous or rectal) shall not be performed nor a general anesthetic given until the patient has had a physical examination including examination of the chest for respiratory infection or cardiac disease and including a urinalysis with tests for albumin and sugar. The results of these examinations, together with the preoperative diagnosis, shall be entered in the patient's record.

G. *Operative records.* An accurate and complete description of the technic of operation and the findings, and a statement of organs or tissues removed, together with the postoperative diagnosis, shall be entered by the surgeon in the patient's record immediately following the operation.

H. *Postoperative care.* After the administration of a general or spinal anesthetic, patients shall be constantly attended by a nurse until they have regained consciousness, or until the effects of the anesthetic have worn off. After any major operation, adequate nursing services shall be provided so that patients are closely watched and given all necessary care.

IV. Regulations for Maternity Hospitals or Services.

All hospitals with maternity service, as defined on page 1 of these regulations, shall conform to the following standards in addition to those specified in the general regulations.

It will be noted that the regulations for maternity hospitals are more detailed and the standards are higher than those applying to other types of hospital service. This is because of the vital role of proper care in the reduction of maternal and infant mortality. Much attention has been given in recent years to efforts to reduce maternal and infant mortality through placing the pregnant woman under medical care early in pregnancy, educating her in the hygiene of pregnancy and the puerperium, and stressing the importance of hospital delivery. Unless maternity hospital standards are kept at a reasonable level, this effort cannot be wholly effective. These regulations are intended, not to hamper the physician with unnecessary red tape nor to dictate to him how he shall conduct his obstetrical work and keep his records, but to aid him by pointing out what are regarded by leading obstetricians and hospital administrators as the essentials of good hospital delivery service and care of the newborn.

A. *Maternity units.*

1. The maternity unit shall be segregated from any medical or surgical service. This shall not preclude the treatment of uninfected medical or surgical cases in separate rooms on a maternity unit.
2. There shall be at least one delivery room, and hospitals with multiple bedrooms or wards shall provide in addition one or more labor rooms.
3. Isolation quarters shall be provided for infected mothers. A mother shall be considered infected if:
   a. She has a communicable disease or is suspected of such, or if she is a carrier.
   b. She nurses an infected infant.
   c. She is delivered outside the maternity unit of the hospital in which she is afterward cared for.
   d. She has an unexplained fever during the puerperium.

4. Labor, delivery room and all other facilities used for infected mothers shall be separate from, and not used for, non-infected patients until they are made safe by cleansing and sterilization.
5. Equipment of a maternity unit shall include a bed-pan sterilizer or individual pans which shall be adequately sterilized for each patient.
6. All nurseries, isolation quarters, formula rooms, examining rooms, labor and delivery rooms, and maternity rooms or wards shall have adequate facilities for the scrubbing of hands, and receptacles for the efficient temporary disposal of soiled linens and waste.
7. Delivery-room technic must be in accordance with recognized and approved obstetrical technic. Equipment shall be such as to make this possible. Oxygen for resuscitation and facilities for blood transfusion shall be available.
8. The advice of an obstetrical consultant should be obtained preceding any major operation or abnormal, induced or complicated labor.
9. Before the removal of the mother or baby from the delivery room each infant shall be labeled with some adequate method of identification approved by the department.
10. The 1 per cent solution of nitrate of silver furnished by the Department of Public Health shall be instilled in each eye of every infant before it leaves the delivery room.
11. Should one or both eyes of an infant become inflamed, swollen and red, or show a discharge, the hospital shall report the case to the local board of health.
12. When such a case is discharged from the hospital, a report in writing shall be sent within six hours after such discharge to the board of health of the city or town to which the infant is to be taken. This report shall state whether the infant is discharged cured or uncured, also the street and number to which the infant is to be taken.
13. Each hospital shall act in conformity with the provisions of Chapter 119, General Laws (Tercentenary Edition), as amended by Chapter 629, Acts of 1941, for the protection of children, and shall not be concerned in, encourage or permit any unlawful disposition of an infant or any arrangement whereby an infant may be deprived of any of his legal rights or be abandoned. The hospital shall not be concerned in placing infants apart from parents or guardian, and if the superintendent knows of any such arrangement he shall at once report the case to the Division of Child Guardianship of the Department of Public Welfare.
14. Upon the discharge of each infant from the hospital, a mark specified by the department, which will serve to identify the infant, shall be placed upon the shirt, diaper, band, dress and other covering.
15. Any neglect or evasion of these rules, or any collusion by the hospital with any person for their evasion, shall constitute sufficient cause for the revocation of the license.

B. *Newborn nurseries.*

1. There shall be a separate nursery for newborn infants, with adequate space, light and ventilation.
2. Bassinets shall be so spaced that they are separated by at least 6 inches on all sides. Suspension of bassinets on double-tier racks shall be prohibited.
3. Isolation quarters shall be provided for infected infants. An infant shall be considered infected if:
   a. It has a communicable disease or is suspected of such, or is a carrier.
   b. If delivered of an infected mother.
   c. If delivered outside the hospital.
4. Common or group carriers for infants shall be prohibited.
5. Cloth or paper covers on a common dressing table or scales shall be changed for each infant.
6. Running hot and cold water and suitable receptacles for the disposal of waste and soiled linens shall be provided in or adjacent to each nursery.
7. Individual sterilized rectal thermometers shall be provided for each infant. All examining instruments should be sterilized before use for each infant. Gauze, cotton and so forth shall be sterilized and kept stored in sterile containers in each nursery.
8. All linens used in the nursery shall be kept separate from those used in other parts of the hospital and shall be laundered separately.
9. All physicians and nurses and any others who must enter any part of the nursery shall scrub their hands thoroughly and put on a clean gown, headgear and mask.

C. *Premature nurseries.* All hospitals with maternity service must provide the minimum equipment listed in this section for the care of premature infants. All standards specified in the section on newborn nurseries must be met in the premature nurseries; in addition, the following provisions shall be made:

1. The premature infant must be cared for in a heated bed or incubator, which must be available in all nurseries.* (In a room with temperature and humidity control designed for the care of premature infants the heated bed is not required.)
2. Some type of heated bed should be ready in the delivery room, in which the infant can be transported to the nursery.
3. Oxygen must be quickly and easily available in both the delivery room and the premature nursery.
4. Suction apparatus should be easily and quickly available in both the delivery room and the premature nursery.
5. The premature should be cared for in a separate premature nursery or should be segregated in the newborn nursery.
6. Isolation facilities must be provided for the infected premature infant.
7. Nurses assigned to the care of premature infants shall not care for infected infants at the same time. Each nurse shall wash her hands for five minutes and put on a clean gown and mask before entering the premature nursery.
8. Visitors must not be allowed in the premature nursery.
9. The premature infant must not be discharged until it weighs more than 5 pounds and its condition is such that it will presumably survive home care.

*A satisfactory incubator can be constructed for approximately $10.00.

10. Before discharge, arrangements for continuous medical supervision by a physician or well-child conference shall be reported by the staff physician to the superintendent.

D. *Formula rooms.* Each maternity hospital shall have adequate facilities for the preparation of formulas for infants — where practical in a separate room. Proper sterilization and refrigeration equipment shall be provided in the formula room or in an adjoining room. Aseptic technic must be maintained in use of containers and utensils and in formula preparation.

E. *Personnel in maternity units and nurseries.*

1. Nursing staff.
   a. Sufficient nurses shall be provided for the adequate care of mothers and infants. These shall be under the supervision, both night and day, of a graduate nurse. After July 1, 1942, all new supervisors shall be graduates of an approved school of nursing and registered in Massachusetts.
   b. Nurses assigned to nursery and formula rooms shall not come in contact with any infected patient or with isolation quarters.
   c. Private nurses must comply with maternity-unit rules and regulations.
   d. No person shall enter the nursery except those immediately concerned with the care of the newborn.
2. All personnel in contact with patients and all food handlers shall be examined on assignment and annually thereafter by a physician designated by the hospital management and shall be certified as showing no evidence of communicable disease. This examination shall include a chest roentgenogram.
3. All personnel shall immediately report any illness, or signs of infection, however slight, and shall be excluded from the maternity unit until seen by a physician.

F. *Visitors and visiting hours.*

1. Visiting hours to maternity services shall be set at such a time as not to coincide with the hours when the newborn infants are in the maternity ward or private rooms for nursing by their mothers. There shall be a minimum of visiting permitted on all maternity services.
2. Children under fourteen years of age shall be prohibited from admittance to any maternity unit at any time.

G. *Records.* Complete and detailed case history records shall be kept of maternity patients and infants. The following outline gives the minimum information which should be included in any hospital maternity record. It is not of itself a record form.

1. Maternal record:

   Patient's name, occupation, age, color, nativity, religion, gravida, address; husband's name and occupation; date of admission and service to which admitted; general history (medical, surgical, Wassermann tests); general physical examination on admission; obstetrical history of each pregnancy, describing prenatal care, month started and regularity, and all abnormalities.

   Obstetrical examination on entrance to service including temperature, pulse, respirations, blood pressure, urinalysis (albumin and sugar), fetal heart rate and abnormalities.

   Labor: a satisfactory description of labor should be recorded, including time of onset, and if induced, reasons therefor; maternal pulse rate during all stages; fetal heart rate. All rectal and vaginal examinations made in any stage of labor must be recorded as to time and findings, and by whom made. Analgesia or anesthesia and medication in any stage should be recorded as to type, amount and time given. In addition, for each stage the following should be recorded:

   First stage: duration, type and frequency and character of pains; rupture of membranes, time, whether spontaneous or artificial; examinations, and anesthesia or analgesia as outlined in above paragraph; fetal heart rate.

   Second stage: duration, examination and anesthesia or analgesia described as outlined above; fetal heart rate; presentation; method of delivery — if operative, reasons; descriptions and names of consultants and operator must be given; description of lacerations; description of any complications.

   Third stage: duration; method of placental delivery; hemorrhage; condition of uterus of mother and infant one hour after delivery. Name of physician who did delivery and assistants.

   Postpartum course: chart of temperature every four hours when awake for five days, then twice daily; if any complication arises, resume four-hour chart; medication and treatment; progress of involution of uterus; type of lochia; condition of breasts and nipples; general condition of mother (including condition of uterus) at time of discharge; date of discharge.

2. Pediatric record:

   Infant's name, sex, color, date and hour born, birth weight and length, period of gestation, mother's name, address.

   Description of complications of pregnancy or delivery; condition at birth, including color, quality of cry; method and duration of resuscitation.

   Findings of initial general physical examination; description of any postnatal disturbances.

   Progress notes; chart of temperature, pulse and respirations at least twice a day if no complications arise; weight chart recording weight at least every other day; condition of cord from day to day; color from day to day; feeding; character and frequency of stools; vomiting; any unusual symptoms.

   Findings of physical examination on discharge, with recommendations.

   Physician's name.

V. Regulations for Tuberculosis Sanatoriums.

All provisions of the general regulations shall apply to tuberculosis hospitals and sanatoriums. In addition, the following special requirements must be fulfilled:

A. *Equipment.*

1. Each hospital, irrespective of its size, shall have x-ray equipment capable of producing satisfactory

diagnostic roentgenograms of the chest and making fluoroscopic examinations of the chest.
2. There must be apparatus for the administration of artificial pneumothorax.

B. *Procedures.*
1. Each patient shall have a medical history taken and physical examination made by a physician within four days of admission.
2. All patients with expectoration shall have their sputum examined for tubercle bacilli by direct smear on admission, and if the smears are negative, more sensitive methods for the detection of tubercle bacilli (concentration tests, cultures or guinea pig inoculation) must be employed. Sanatoriums with less than 50 beds may arrange to have a laboratory, approved by the department, perform these tests. All patients who are raising sputum, with the exception of progressive far-advanced cases, should have their sputums examined at least once a month.
3. All patients shall have a chest roentgenogram on admission and as often thereafter as is necessary to detect changes in the pulmonary lesions, with a maximum interval of once in four months, and within two months of discharge.
4. Patients shall be provided with cardboard sputum boxes and paper handkerchiefs or satisfactory substitutes therefor, which shall be destroyed by incineration after use. Reasonable precautions shall be taken against exposing personnel and other patients to infection from sputum-positive cases.

C. *Personnel.*
1. Each hospital shall have either on its resident or visiting staff at least one properly qualified tuberculosis specialist.
2. Whenever indications exist, examination and treatment by a consulting nose and throat specialist shall be provided. Arrangements shall be made for the performance of bronchoscopy whenever, in the opinion of the staff, this procedure is indicated.
3. The services of a dentist shall be provided regularly and as needed.
4. All personnel regularly employed in the hospital who come in contact with patients shall have a roentgenogram of the chest at least annually.

BOOK REVIEWS

Essentials of Endocrinology. By Arthur Grollman, Ph.D., M.D. 8°, cloth, 480 pp., with 74 illustrations. Philadelphia: J. B. Lippincott Company, 1941. $6.00.

This book is intended as a broad survey of the entire subject of endocrinology for the "average medical reader," and serves as a good up-to-date summary of this rapidly developing field.

The author is primarily a pharmacologist and has been a chemist and physiologist. It is not surprising, therefore, that his approach is that of the laboratory experimenter rather than that of the clinician.

After an introductory chapter, the author takes up each endocrine gland in turn, grouping together the glands of the cranial cavity, the branchiogenic organs, the abdominal endocrines, the hormones of the reproductive system and the hormones derived from nonendocrine organs. In discussing each gland, he gives a rather detailed account of its embryology and histology, and draws heavily on the available data from comparative anatomy. The physiology and the principal experimentally proved facts are concisely presented, whereas all theories and ideas not conclusively proved experimentally are given short shrift. Many widely held notions are dismissed as without valid foundation. Thus, the conception of the pituitary gland as secreting numerous separate hormones "is undoubtedly erroneous, and future investigation may reveal that the hypophysis elaborates a single hormone which is responsible for all of the manifestations of the gland."

The clinical aspects of endocrinology, including therapy with the available preparations, are by no means neglected; yet they are given relatively less consideration than the "average medical reader" might wish. There are a few statements that might be questioned. For example, in the treatment of myxedema, the author states that it is "safe to initiate therapy with a daily dose of 2 grains [of thyroid]." In discussing the causes of destruction of the anterior portion of the pituitary gland in Simmonds's disease, he states that "the most common one is emboli originating usually in the parturient uterus which occlude the terminal arteries of the anterior hypophysis."

On the whole, this is a well-written, critical textbook. There are well-chosen references at the end of each chapter, a good index, and a table, in the front and back linings, of some commercially available endocrine products and their standardization. Unfortunately, there are numerous typographical errors.

The Biologic Fundamentals of Radiation Therapy. A textbook. By Friedrich Ellinger, M.D. With a preface by Maurice Lenz, M.D. English translation by Reuben Gross, M.D. 8°, cloth, 360 pp., with 79 illustrations. New York: Elsevier Publishing Company, Incorporated, 1941. $5.00.

The book covers more ground that its title implies. In addition to the biologic fundamentals, it also delves into the underlying principles of electrophysics that pertain to this subject. Although the part of the light spectrum that is therapeutically most important will remain invisible to the *physical* eye of the reader, this book will enable him to see it with his *mind's* eye.

The introduction describes the electromagnetic spectrum and the combination of the quantum and wave theories of light. A section on biologic considerations or the action of radiation on living matter follows. This leads to discussions of absorption of radiation by the respective medium and of the effective quantum energy as a function of wave length at the site of absorption. In the light of these and similar basic principles, roentgen-rays, the gamma radiation of radium, the Grenz rays (ultrasoft roentgen rays), ultraviolet rays and the action of visible light are successively treated.

The technic of application of these energies to the various tissues, organs and systems of the body is discussed in the light of the previously mentioned physical principles and biologic effects.

Important factors, beneficial and injurious, that are manifested by the local reaction of the skin, the portal of entry of these rays, and their indirect effects on the deep-seated structures of the body are taken up in a logical and scientific manner.

The author is as honest in his statements of fact and hypothesis as he is profound in his discussion of the underlying theory. He is as generous in the details of technic of application as he is broad in his consideration of the various physiologic and pathological problems that confront the physician in his practice of medicine. The reviewer therefore heartily endorses this work to the student, general practitioner and specialist alike. Radiation therapy, particularly that employing the ultraviolet ray, has been used widely but not always wisely. This treatise may help to remedy the situation.

(Notices on page x)